FLUORESCEIN ANGIOGRAPHY OF POSTERIOR FUNDUS IN MYOPIA

THESIS

FOR

MASTER OF SURGERY

(OPHTHALMOLOGY)

BUNDELKHAND UNIVERSITY

JHANSI (U. P.)

1989

SATISH CHANDRA

C E R T I F I C A T E

This is to certify that the work entitled. "FLUORESCEIN ANGIOGRAPHY OF POSTERIOR FUNDUS IN MYOPIA" which is being submitted for thesis of M.S. (Ophthalmology) by DR. SATISH CHANDRA has been undertaken by the candidate under my supervision and guidance. He has carried out the work independently and his observations were periodically checked by me.

He has also completed the required period of stay in the department.

(G. D. GUPTA)
M.S., D.O.M.S.
Head,
Department of Ophthalmology,
M.L.B. Medical College, Jhansi.
(SUPERVISOR)

Dated: 8th Sept. 1968.

C E R T I F I C A T E

This is to certify that the work entitled, "FLUORESCEIN ANGIOGRAPHY OF POSTERIOR FUNDUS IN MYOPIA" which is being submitted for thesis of M.S. (Ophthalmology) by DR. SATISH CHANDRA has been under taken by the candidate under my supervision and guidance. He has carried out the work independently and his observations were periodically checked by me.

He has also completed the required period of stay in the department.

(B. S. JAIN)
M.S.
Lecturer,
Department of Ophthalmology,
M.L.B. Medical College, Jhansi.
(CO-SUPERVISOR)

Dated: 8th Sept. 1988.

A C K N O W L E D G E M E N T S

The over whelming feeling of gratitude and indebtedness, which conceived in my mind during the entire phase of this work try hard to take the shape of words when I start to pen acknowledgements.

It start with deep sense of gratitude to my esteemed and exalted teacher Dr. G.D. Gupta, M.S., D.O.M.S., Head, Department of Ophthalmology, M.L.B. Medical College, Jhansi for his constructive and valuable suggestions and reasoned criticism and his invaluable guidance & keen interest in my work, has been a source of constant inspiration and consistent enthusiasm to me through out the period of study. His vigilent supervision, meticulous attension to details at the expense of busy socio-professional and personal life have gone a long way towards the success of this work.

I am highly greatful to Dr. B.S. Jain, M.S., Lecturer, Department of Ophthalmology, M.L.B.Medical College, Jhansi under whose able guidance and supervision. I worked on this thesis and from whom I received not only inspiration but also clear insight into the subject. His sincere and unlimited help went to long way in the completion of this work.

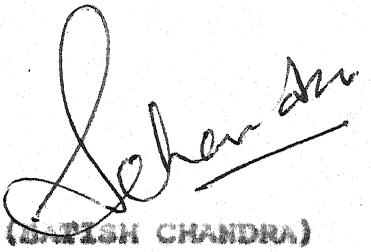
I acknowledge with sincere thanks to Dr. V.K. Misuriya, M.S., Lecturer, Department of Ophthalmology, M.L.B. Medical College, Jhansi for his helpful suggestions and day to day inecourgment with a brotherly attitude.

Though the words of appreciation are not needed for friendes and calleagues but the way Dr. S.K. Pande, Dr. Ravindra Kumar, Dr. Gireesh Sharma, Mr. D.C. Singh & Mr. Amar Singh helped me in various ways in building up the infreestructure of this study, is unforgettable and with out whom this work would have been a lot more difficult.

I am expressing my sincere thanks to the patients who were subjected the examination for this study without which this work could not have been carried out.

I have no words to express my gratitude to my wife Sneha for her constant moral support in the dreaded days of over work and for extending a help ful hand beyond all limits and expectations.

Last but not the least I take the oppertunity to acknowledge greatfully, the debt I owe to my parents and family members for all that they have done for me.



Dated: 08th Sept. 1988. (BRIJESH CHANDRA)

C O N T E N T S

*****
***
*

INTRODUCTION

INTRODUCTION

Fluorescein angiography of the fundus is relatively a newer technique to study the dynamic vascular aspect of the choroid and the retina.

Chao and Flocks described fluorescein angiographic technique in as earliest as 1958. Since the clinical introduction of this technique by Novotny and Alvis (1961), fluorescein angiography has been widely used for better visualization, understanding and documentation of various intraocular disorders.

It is wall established observation that the eye with high myopia has a poor visual acuity due to wide spread degenerative changes in the fundus. In the simple myopia fundus may be normal or it may have minimal changes in the form of simple myopic crescent, while in pathological type of myopia wide spread chorio-retinal degeneration, pigmentary desepration, localized pegmentary clumps and irregular diffused or localized retinal pigment epithelium defects are the important ophthalmoscopic findings (Duke Elder 1970, Curtin and Kartin 1971), still no satisfactory

explaination is available to account for the development of such degenerative changes in the myopic eyes.

Degenerative changes in pathological myopia are due to stretching as a consequence to increased anteriopesterior diameter. It is, however, a common observation that degenerative changes are not constant in all cases with similar referative errors, therefore, it becomes more important as to why only certain eyes are Prone to such effects of stretching.

Recently a few workers have tried to correlate the occurance of degenerative changes in highly myopic eyes with fluorescein angiography (Taldaeva and Khasanova 1975, Stanges et al. 1976, Yoshihara et al. 1976, Levy et al. 1977, Baizhanova 1978, Avetisov E.S. and Savitskaya 1977, Giovannini et al. 1980, Hoyt C.S. et al. 1981, Michael Shapiro and Suresh R. Chandra 1985). The results are so incouraging that changes hitherto described purely due to stretching caused by vascular phenomenon. However, the description is inadequate.

With this view, the present study has been planned mainly to study the vascular changes in myopia.

Accordingly, the following aims and objects were set up for the study:

To perform fluorescein angiography in normal individuals and patient with simple and pathological myopia and to describe the fluorescein angiographic changes in such eyes.

To correlate the findings so obtained on fluorescein angiography in myopia with the ophthalmoscopic appearence.

To study the choroidal circulation in normal and myopic individuals.

The study has been conducted in 38 eyes of 26 individuals and an attempt has been made to describe the extent and pathogenesis of degenerative changes in myopia.

REVIEW OF LITRATURE

REVIEW OF LITERATURE

I- FLUORESCEIN FUNDUS ANGIOGRAPHY AND ITS RECENT MODIFICATION:

Fluorescein angiography is relatively a newer technique for the study of dynamic aspects of the retinal and choroidal circulation and associated disorders located at particular areas in the fundus. Novotny and Alvis (1961) introduced this technique for the first time in ophthalmic practice. Since then a number of technical advancements have been made in this field. A number of workers have described the clinical utility of this procedure in various ocular disorders (Dollery et al, 1962; Ferrer, 1965; Allen et al, 1966; Hodge and Clement, 1966; Haining, 1966; O'day et al, 1967 and Hayreh, 1968). A lot of experimental work has also been done on monkeys to know the physiological anatomy of the choroidal and retinal vasculature and the associated pathological entities (Hayreh, 1969, 1970, 1973, 1975; Hayreh and Baines, 1972, 1973; Oosterhuis and Boen-Tan, 1971 and Hyvarinen et al, 1969). Recently various workers studied the evaluation of various lesion in myopia. Je Rona M. Levy, Henry M. Pollock & Miriam J. Curtin (1977), Fried, M., Stubert, G. A., Meyer-Schwickerath & Wessing A., (1980), Giovannini, A. & Colombati, S.,

(1980), Giovannini, A., Colombati, S. & Ciliberti, C. (1980), Hoyt, C.S., Stone, R.D. & Fromer, R.C. et al (1981), Michael, Shapiro & Suresh, R. Chandra (1985).

Usually the fluorescein fundus angiography of both eyes is done in separate sittings but binocular fundus fluorescein angiography simultaneously of both eyes also has been described (Hisatomi and Suzuki, 1974; Kooijman, 1972; Laum, 1976). Most of the workers prefer to take fluorescein angiograms in black and white while some of them have also tried to take coloured angiophotographs (Shikano and Shimizu, 1968; Hendrickson et al, 1970). The fluorescein angiography of the equator and beyond is also possible with the introduction of 100° camera (Pomerantzeff and Govingson, 1971) and equator plus camera (pomerantzeff, 1975) as with the conventional equipment, angiography gives best results of the central fundus only.

DYE:

A number of chemical dyes have been used for the purpose of fluorescein angiography but the sodium fluorescein dye is regarded as the best for this purpose. It was synthesized by Von-Beyer (1871) and was used in ophthalmic practice by Paul Ehrlich (1881). It is a water soluble low molecular weight compound. Its

molecular weight is 376.24 and it gives a maximum fluorescence at pH of 7.4 . About 40-80% of it is bound to plasma proteins when injected into the blood. It has got no firm bond with the vital tissues of the body and is therefore excreted in about 24-36 hours after intravenous injection but it stains skin and mucous membrane yellow for about 2-4 hours and causes yellowish discolouration of the urine for 1-2 days. The dye has got remarkable fluorescent properties and the conversion of absorption light to fluorescent light is almost 100%.

The maximum light absorption and excitation of fluorescein is between 480-510 nm whereas the emission curve for fluorescein has a maximum between 500-530 nm. It diffuses freely in the eye through choriocapillaris, bruch's membrane, optic nerve and sclera but not the healthy retinal vessels, retinal epithelium and larger choroidal vessels. The compact endothelial cells of the retinal vessels and compact retinal pigment epithelial cells act as barriers to the passage of fluorescein (Schatz et al, 1978).

The sodium fluorescein dye has been given by catheterization into the big veins in experimental animals (Mayrch, 1968; Dollery et al, 1962). But the dye is conventionally given by intravenous route into the antecubital veins in human beings. Since the circulation in the antecubital vein is very slow and the dye undergoes

dilution of about 600 times before reaching the ocular vessels, the use of 5-15 ml of normal saline immediately after it has been advised by Flower (1973). Depending upon one's personal choice and experience, various concentrations and dosage schedules have been advised. It can be given as 5% - 5 ml (Charamis, 1966; Novotny and Alvis, 1961); 5% - 10 ml (Allen et al, 1966); 20% - 5 ml (Oosterhuis and Lammens, 1965); 25% of - 3 ml (Archer , 1972). Rosen (1969) prefers to give it in doses of 10 mg/kg body weight.

Practically the dye has been found to be much safer. Of all the side effects, nausea and vomiting are the commonest and occur in about 5% of cases (Hayreh, 1974). Severe reactions include anaphylactoid reactions of various types (La Piana and Penner, 1968). Stein and Parker (1971) analysed the side effects in about 55 cases and main side effects were seen in the form of urticaria, allergic reactions of skin, laryngeal oedema and hypotension. Shock, fatal myocardial infarction and cardiac arrest are rare. Other rare side effects like achromatopsia (Archer, 1972) and hemiplegia (Mathews et al, 1979) have also been described.

In addition to sodium fluorescein dye, a relatively new dye Indocyanine green (ICG) has been used for the better understanding of the choroidal vasculature

(Kogrue and Chromokos, 1969; Kogrue et al, 1970; Flower, 1972; Flower, 1973). It is rapidly excreted in the bile without any recirculation in the fundus. It has a peak emission at or near 800 nm (Archer, 1972) and is not masked by retinal pigment epithelium. It has also been used in combination with sodium fluorescein (Flower, 1973). Lissamine rhodamine - B (Machemer , 1979), acridine dye (Kuwamoto, 1969), riboflavin dye (Shimotori and Aoki, 1975) are other dyes having fluorescent properties but less marked than sodium fluorescein dye. These have been used in experimental animals only.

2. FILTERS

One of the important prerequisites for fluorescein angiography is the choice of proper and matched dual filter system. The barrier filter and exciter filters should be well matched to avoid the problem of pseudofluorescence. Pseudofluorescence is a term for non fluorescent light that passes through the dual filter system and results in artificial fluorescence (Schatz et al, 1978). The exciter filter is ideal which removes all the wavelengths from the incident light except in the range of 480-510 nm wavelengths which is the absorption peak of fluorescein excitation. Similarly an ideal barrier filter should cut off all the wavelengths except those in the range of 500-530 nm which is the fluorescent or emitted peak of fluorescein. There

should be no overlap between the filter curves. To test for the correctly matched filter combination, a control picture of the fundus is taken with the filters in the position prior to the injection of fluorescein dye. This should show a completely blank picture without any fundus details(Hayreh , 1974).

The first filter system which minimizes the pseudofluorescence was reported by Hodge and Clemett (1966). Later on Baird atomic interference filters B_4 and B_5 (Haining and Lancaster, 1968). B_4 in conjunction with Kodak wratten 12 (Machemer et al, 1970). Broad band filter system (Frazier and Allen, 1972). Spectrotech, SB_4 and SB_5 interference filters (Mendiros, 1974) and De Lori filter system (Delori and Ben-Sira, 1975) were described. These are well proven good quality filter combinations.

3. STEREOANGIOGRAPHY

The first step towards improvement of fluorescein angiography was undertaken by Allen et al (1966) who introduced the concept of stereoscopic angiography. In this procedure, consecutive stereoangiograms are taken employing a three dimensional picture of the fundus. For this purpose, Donaldson (1964) devised a special fundus camera which had many advantages in the form of higher magnification, acute focussing mechanism and reflex mirror system. The introductio of Allen's stereoseparator and other technical advancements have helped in getting rapid sequence stereo-angiophotographs

(Allen et al, 1966; Harel et al, 1968; Crock, 1970; Haining and Lancaster, 1968).

4. FLUORESCEIN CINE-ANGIOGRAPHY

Fluorescein cineangiography first introduced by Hart et al (1963) and further modified by Eberhoff et al (1965) is one of the other technical advancements in the field of fluorescein angiography. With this procedure the retinal circulation could be easily studied and documented cinematographically. Hyvarinen and Nieminen (1967) modified the Zeiss fundus camera for fluorescein motion picture angiography of rabbits. The advantages of this method were a large field and a high exposure rate of 24/second. This is very useful in studying the rapid arterial filling in the individual frames. Further, advancements in the field of cineangiography have been described by Hill and Young (1976).

5. TELEVISION FLUORESCEIN ANGIOGRAPHY

Another milestone in the field of angiography was the development of television fluorescein angiography. The first model for television fluorescein angiography invented in 1968 was quite complex (Van-Heuven et al, 1971). The method consisted of utilising an image orthicon television camera, a videotape recorder and Zeiss fundus camera. It proved to be very helpful in permanent documentation and accurate reproduction of dynamic aspects of retinal vascular flow. In the next four years from 1969-1972 various

modifications were made utilizing a number of camera tubes secondry electron conduction, silicon matrix and silicon intensifier target types. Yuhasta et al (1973) introduced the videotape recording system for the teaching purposes. Later on stereoscopic TV-angiography was developed (Von-Heuven and Schaffer, 1973; Van-Heuven and Schaffer 1974). Shimizu (1976) introduced a new TV-guided camera in which the infrared light was used and this is used in the dark room in undilated pupils. The latest developments in the field of TV fluorescein angiography is the introduction of solid state imaging devices and direct computer processing of the retinal images and as such the better under-standing of retinal haemodynamics has been described (Pehman et al, 1976).

6. IRIS FLUORESCEIN ANGIOGRAPHY

Anterior segment fluorescein angiography is comparatively a new advancement in the field of angiography and was first utilized by Jensen and Lundbaek (1968) in recent and long term diabetics. The role of iris angiography is emphasized in various diseases affecting the iris tissue and its vascularity (Vannas, 1969). Jensen (1973) described a new model for fluorescein angiography of both irides similtaneously in single sitting.

Fluorescein angiography is a very useful tool for the better understanding of the normal and abnormal

haemodynamics of the choroidal and retinal circulations and the associated disorders. As soon as the dye is injected into the vein it reaches the fundus very quickly in about 9-12 seconds (Archer, 1972). The time taken for the dye to reach the central retinal artery in the disc area is known as arm to retina circulation time which depends largely upon the concentration of dye and its route of administration. The dye further passes through the retinal arteries, artrioles, capillaries, venules and returns to the central vein. The flow in the vein is first laminar and later the veins are completely filled up. After sometime the recirculation of dye occurs and the fluorescence gradually diminishes till the whole of dye has been washed out the circulation. It has been confirmed that the flow to choroid is earlier than the retinal vessels (Evans et al, 1973; Shimizu et al, 1974).

The results of fluorescein angiography largely depend upon the different spectrum distribution factors like an emission flash tube, transmittance of the exciter and barrier filters, activation spectrum, fluorescence spectrum of the fluorescein in the blood plasma, reflectivity of the retina and the film speed. However, from time to time modification in the procedure and use of dye have been undertaken for better results of the angiograms and retinal and choroidal haemodynamics.

II- RELEVANT ANATOMY

I. DISC CIRCULATION

The optic disc circulation has been studied in detail by Hayreh (1969, 1972, 1974) and Ernest and Archer (1973). Hayreh (1969, 1972) has divided the optic nerve head into four parts depending upon its vascular supply as observed after fluorescein fundus angiography.

(i) Lamina Cribrosa

It is a dense compact connective tissue which is continuous with the sclera with many openings in it for the transmission of the nerve fibre bundles. It is supplied by centripetal branches from the short posterior ciliary arteries either directly or through the arterial circle of Zinn.

(ii) The Prelaminar region

The part of the disc in front of the lamina cribrosa is known as the prelaminar region. Here the connective tissue of the lamina cribrosa is replaced by the loose glial tissue. This is attached peripherally to the choroid and bruch's membrane. The region is mainly supplied by the centripetal branches from the surrounding peripapillary choroidal vessels. Possibly, this part may also get some branches from the short posterior ciliary

arteries. Central retinal artery does not usually contribute to the vascular supply of this part.

(iii) Surface nerve fibre layer

It is the most superficial layer, consisting of compact nerve fibres. This is continuous with the nerve fibre of the retina and is covered by internal limiting membrane. This region is supplied by the branches of the central artery and sometimes the temporal part of this layer receives a contribution from the peripapillary choroid.

(iv) Retrolaminar region

This part is supplied mainly by the centripetal branches from the pial-vessels which are mostly the branches of peripapillary choroid and circle of Zinn. Three quarters of the nerve may be supplied by the centrifugal branches of the central retinal artery.

Venous drainage

The optic nerve head and the retrolaminar part of the optic nerve are drained by the central retinal vein. The prelaminar region is drained into the choroidal veins. There are no venous channels corresponding to the circle of zinn. That there is a communication between the central retinal vein and the choroidal circulation in the prelaminar region has been

confirmed after a complete retinal vein occlusion behind the lamina cribrosa.

RETINAL CIRCULATION

Central retinal artery and its branches supply the whole of the retina. The central retinal artery originates from the ophthalmic artery but occasionally may arise from a ciliary artery(Wyber, 1956). The central retinal artery divides and redivides to supply the temporal and nasal halves of the retina both superiorly and inferiorly through its various branches. The larger retinal arteries and the pre-capillary arterioles are located under the internal limiting membranes in the nerve fibre layer. The retinal capillaries (along with the post-capillary venules) are located deep in the inner nuclear layer (Schatz et al, 1978). In this way the portion of the retina extending from the inner nuclear layer to the internal limiting membrane is supplied by the retinal blood vessels in two separate planes. The outer half of the sensory retina and the retinal pigment epithelium are not supplied by the retinal blood vessels. The retinal arteries are the end-arteries. The venous drainage of the retina is ultimately through the central retinal vein.

CHOROIDAL CIRCULATION

Earlier it was believed that the choroidal vasculature is a continuous bed with no segmental distribution (Nicholls, 1938; Wyber, 1954). Fluorescein fundus angiography has completely changed this old concept and now it has been confirmed that the choroid has got a segmental nature of vascular supply (Hayreh, 1969; 1973; 1974; 1975; Hayreh and Baines, 1972; Oosterhuis and Roen-Tan, 1971; Anderson and Davis, 1976).

(1) ARTERIAL SUPPLY OF CHOROID

The choroid is supplied by posterior ciliary artery and its subdivisions which are as follows:

(a) Main posterior ciliary arteries

There are two main posterior ciliary arteries. The lateral and medial posterior ciliary arteries supply the temporal and nasal choroid respectively. Sometimes superior posterior ciliary artery has also been noticed (Hayreh, 1976).

(b) Short posterior ciliary arteries

They are about 10-20 in number and are the subdivisions of main posterior ciliary arteries. They supply small sectors varying in size, shape and location. Smaller subdivisions of short posterior ciliary arteries supply still smaller segments of the choroid.

(c) Long posterior ciliary arteries

These are usually two in number and are derived from the ophthalmic artery. They pierce the sclera slightly farther away from the nerve in the horizontal meridian one on the nasal and the other on the temporal side. In contrast to the classical description that these arteries supply the part of the choroid in front of the equator of the eye, fluorescein fundus angiography has revealed that these supply a narrow segment of the choroid extending forwards from its point of entering into the globe(Hayreh, 1973).

(2) CHORIOCAPILLARIS

Fluorescein fundus angiography has revealed that each terminal choroidal arteriole joins a choriocapillary in the centre of a segment. The venule draining this segment lies around the periphery of this segment (Hayreh, 1976). Various segments are an independent units with no communications among themselves and are arranged like a mosaic.

(3) CHOROIDAL VENOUS DRAINAGE

Venous drainage of the choroid is through four vortex veins which have a segmental distribution with poor communication between the adjacent veins(Hayreh and Baines, 1973). Finally the blood is drained into the

ophthalmic veins.

Interrelationship between choroidal and retinal circulation

The ciliary circulation is the main source of the blood supply to the prelaminar, laminar and retrolaminar parts of the optic nerve. The main retinal vessels supply the surface layer of the optic disc. It is well known that the external retinal layers are supplied with the choroidal capillary layer of the choroid and hence any pathology of the choroidal vasculature may lead to the retinal changes (Balshanova, 1978).

III-FLUORESCEIN FUNDUS ANGIOGRAPHY

Fluorescein fundus angiography is a useful tool for understanding the normal vasculature of the optic disc, choroid, retina and various associated lesions. Various quantitative aspects of circulation can be known with the help of this method.

As soon as the dye is injected into the antecubital vein it reaches the systemic circulation and profuses in the eye through the central retinal artery and the ciliary vessels.

A. Transit of the dye:

After few seconds of the injection the dye passes into the choroidal and retinal circulation, the former a bit earlier than the latter(Evans et al, 1973; Shimizu et al, 1974; Archer et al, 1970). From the central retinal artery the dye passes into its branches, the arterioles, precapillary arterioles, capillaries and is returned to the venule, veins and finally into the central retinal vein.

Dollery et al (1962) divided various phases of dye transit into (i) choroidal flush, (ii) early arterial phase, (iii) late arteriolar phase, (iv) capillary phase, (v) early venous phase, (vi) and late venous phase. Novotny and Alvis (1961) divided it into arterial phase, arterio venous phase and venous phase only. Hayreh(1974) adopted a different terminology for the same as pre-retinal arterial phase, retinal phase, retinal arteriovenous phase, retinal venous phase and venous phase about 10-15 minutes after the injection of the dye.

B. Normal disc fluorescence

Fluorescein fundus angiography has revealed that the filling of the disc occurs early before the dye reaches the central retinal artery. This again confirms that the main vascular supply of the disc is through ciliary vessels. Moreover the filling of the disc occurs

earlier on the temporal side which shows that this part is more vascular than the nasal part, though it appears to be comparatively pallor on ophthalmoscopic examination (Hayreh, 1969, 1972, 1974).

The prelaminar fluorescence that is of choroidal origin remains throughout and increases in intensity in the late venous phase of the dye transit. In the early and late phase, pre-laminar fluorescence is the only source of the fluorescence. In the late phases this becomes intensive because of leakage of dye from the choroidal capillaries of prelaminar origin. In the arterial and arteriovenous phases, the surface layer fluorescence (that is of retinal origin) contributes maximally. The fluorescence of the optic disc is usually maximal in the early arteriovenous phase of the retinal circulation and coincides with the maximal background fluorescence of the fundus (Hayreh, 1969), when a cilioretinal vessel is present this originates from the annulus of zinn and enters the fundus at the temporal disc margin with a sharp bend. In view of their origin from the posterior ciliary arteries, they are indicators of choroidal circulation(Weesing, 1969). Filling of the cilioretinal artery precedes filling of central retinal artery(Archer et al, 1970; Hayreh, 1974). The incidence of cilioretinal artery has been found to be 4.9% (Justice et al, 1976).

C. Disc fluorescence in myopia and related disorders

Abundant deep disc capillaries which are not seen ophthalmoscopically can be seen on fluorescein angiography in high myopes. These are found to take origin from the arterial circle of Zinn-Haller and juxta papillary choroidal vessels and even in presence of juxtapapillary chorioretinal atrophy the circle of Zinn-Haller is clearly demarcated in the fluorograms (Yoshihara et al, 1976). Further Yoshihara et al(1976) also presumed that the reactive glial proliferation covering the vascular nets of the optic disc was responsible for the change in colour of the optic disc and on fluorescein angiography, faint residual fluorescence could be observed inside the disc in late phases. They concluded that the change in the colour of disc as seen by ophthalmoscopy did not reflect its vascularity in myopic eyes.

In myopic eyes not associated with glaucoma, the filling of the disc is complete. When associated with chronic simple glaucoma, there is invariably a filling defect of various degree depending upon the extent of involvement of the disc by the glaucomatous process (Tolusson and Schwartz, 1977). In cases of primary optic atrophy, there occurs a reduction in the deep network of ciliary and choroidal capillaries. Therefore, in the arterial phase of angiography the loss of surface background fluorescence has been observed (Archer, 1972).

D. Disc crescent

One of theimportant fundus findings in high myopia is the presence of a crescent which is known as myopic crescent or conus. The choroidusually terminates some distance from the margin of the disc and is completely or partially absent in the area of the crescent.The outer retinal layers and the pigment epithelium may be absent in the area of the crescent (Duke Elder, 1970).

The cause of the myopic crescent is disputable It may be absent in high myopia and may be present in lesser degree of myopes. It is essentially an atrophic - phenomenon and not merely due to stretching. The choroid is atrophic in the area of crescent (Parson, 1978). In cases of myopic crescent the peripapillary choroid shows plexus like appearance in the crescent area. In some cases a cilioretinal artery has been observed arising from this plexus (Stangos et al, 1976). The presence of a crescent is revealed in both early and late phases of fluorescence. The early fluorescence of the crescent is distinct from disc fluorescence of the crescent is distinct from disc fluorescence (Rosen, 1969). In the late phases the choroidal leakage combined with the staining of sclera which forms the floor of the crescent causes the hyper fluorescence of the crescent (Rosen,1969).

E. Arm to retina circulation time

The time taken by the dye to reach the major retinal vessels at the disc after the injection is termed as arm to retina circulation time. This has been found to vary between 8-14 seconds (Archer, 1972) 8.0 ± 1.0 second (Shimizu et al, 1974), but 10-12 seconds in younger and 10-15 seconds in old individuals (Schatz et al, 1978). The choroidal filling is earlier than that of the retinal vessels by about 1.5 seconds (Evans et al, 1973), 0.8 ± 0.4 seconds (Shimizu et al, 1974) and 1 second (Krill, 1970). Arm to retina circulation time has been calculated by various workers by serial fluorescein angiography and cinematography and results are variable (Wessing, 1969). Arm to retina circulation time has been found to be longer in high myopes (Yoshihara, 1979) and normal in simple myopia (Ferrer, 1974) while it has been found to be markedly longer in cases where glaucoma is associated with myopia (Ferrer, 1974).

F. Retinal fluorescence

As soon as the dye reaches the major retinal vessels of the disc, it rapidly flows into its branches. Various phases of transit of dye are as follows:

(i) Retinal arterial phase

The peak fluorescence of the arteries is

gradually attained within 1-2 seconds (Archer, 1972) or 2-3 seconds (Dollery et al, 1962). This is termed as arterial phase of the dye transit. The temporal arteries fill slightly in advance of the nasal retinal arteries. The macular arteries fill very rapidly. Oberoff et al, (1965) documented the retinal circulation time cinematographically and found it to be 1.2 seconds, macular; 1.8 - 1.9 seconds total temporal; 2.6 - 3.0, temporal without involving macula; 2.3 - 2.4 second - nasal retinal vessels. The diameter of the retinal arteries and veins at disc margin is 100-110 μ and 130 μ (Wessing, 1969). & the capillary diameter has been found to be 3.5 - 6 μ respectively (Spitznas, 1976 a). In fluorescein angiography the retinal vessels appear to have wider lumen than in the standard retinal photographs (Allen et al, 1966; Shikano and Shimizu, 1968). This is because, the fluorescein stains the normally transparent plasma between the vessel wall and the central core of the erythrocytes (Hayreh, 1974).

(ii) Retinal capillary phase

The filling of the retinal capillaries immediately follow the retinal arterial phase of angiography. Peak fluorescence of retinal capillary bed occurs during the mid retinal venous phase of angiography and fades rapidly during the late venous phase (Archer, 1972). Four main types of capillaries are observed after fluorescein angiography.

(a) Epipapillary capillaries

They run radially from the larger arterioles over the disc surface to supply the peripapillary retina. They are more marked on the temporal side of the papilla.

(b) Peripapillary capillaries

They lie adjacent to the optic disc and extend in an arcuate fashion.

(c) Macular capillaries or perifoveal capillaries

These capillaries show the maximum fluorescence after 12.4 - 30 seconds for a 10% fluorescein solution and 15.3 - 27.7 seconds for a 25% solution with a mean of 21.7 ± 5.7 seconds and 20.1 ± 6.8 seconds respectively (Evans et al, 1974). The individual capillaries canbe identified in the perifoveal region.

(d) Peripheral capillaries

Towards the periphery, the capillaries become less marked and may not be even visualised in the region of orgaserrata. The filling of the capillaries tend to be patchy at first which soon becomes generalised and diffused.

(iii) Retinal venous phase

The smaller venules at the macula fill first. As the fluorescein enter the veins horizontally from the venules, the flow appears to be laminar. The vascular flow

is faster in the centre of the blood stream than on the sides, causing the laminar pattern of retinal venous flow. As many as seven laminas may be noted in the blood stream of a major retinal vein. The presence of several laminar columns in the major veins denotes the mid-retinal venous phase (Archer, 1972). Later on they become intensive and are completely filled in next 5-10 seconds (Schatz et al. 1978). The first high concentration flush of fluorescein begins to empty from the retinal and choroidal circulation in approximately 30 seconds after injection of dye(Schatz et al, 1978). In normal retina, the choroidal and retinal vasculature slowly empties of fluorescein in about 3-5 minutes after injections. In about 10 minutes after injection there remains no fluorescence in the vessels and only the late staining of sclera and bruch's membrane may be seen (Schatz et al, 1978). The peak fluorescence in the retinal artery and in the retinal veins can be observed by densitometry (Hickam and Frayser, 1965). Using a time concentration curve of fluorescein from successive photographs Hickam and Frayser (1965) concluded a mean of 4.7 second ± 1.1 second for the dye to pass the arterial and venous points in the circulation at the posterior pole of eye.

G. Retinal fluorescence in myopia and related disorders

Ratio of artery to vein calibre has been noted to be 1:1.36 as compared to 1:1.25 in the normal eyes. The diameter of the vessel decreases with the progress of myopia and this explains the occurrence of dystrophic changes in

the retina in myopes(Khasanova and Taldaeva, 1975). It has also beenoticed that there is a lengthening of both the arterial and venous phases of transit of dye in myopic eyes(Khasanova and Taldaeva, 1975) rich superficial retinal capillaries are clearly visible on the surface of the disc and are seen to extend towards the macula in the myopic eyes (Yoshihara et al, 1976).

N. Choroidal fluorescence

xperimental occlusion of posterior ciliary arteries in rhesus monkeys produced a patchy fundus lesion in the region of the supply by the occluded artery. It has been studied on fluorescein fundus angiography that the occlusion of lateral posterior ciliary artery produced the extensive lesions compared to the medial posterior ciliary artery occlusion. (Hayreh and Baines, 1972). Normally the retinal pigment epithelium masks the choroidal vasculature due to its fuschin and Xanthophyll pigments, more so in the macular region (Schatz et al, 1978). Since the pigment epithelium and the Xanthophyll both have absorption spectra in the range of fluorescein absorption Peak at 490 nm) and the pigment epithelium also has an absorption spectrum in the range of fluorescein emission (Peak at 520 nm), their presence in eye hinders the visualization of underlying fluorescein filled choroidal vessels (Flower, 1972). However, the normal flow of choroidal circulation has been studied in myopic and albinotic fundus with defective pigment epithelium

(Archer et al, 1970).

Fluorescein fundus angiography has helped in understanding of the watershed zones, which are the important vulnerable zones in the choroid(Hayreh, 1973, 1974, 1976). Watershed zone is formed by the border between any two neighbouring vessels from the main posterior ciliary artery right down the terminal choroidal vessels. Macular region is the meeting point of many watershed zones of the various short posterior ciliary arteries and watershed zones of all the vortex veins(Hayreh, 1974). The watershed zones between the short posterior ciliary arteries and the anterior ciliary arteries are located in the peripheral part of the choroid (Hayreh, 1976).

Arm to choroid circulation time

The arm choroid circulation time is shorter than arm is retina time and is found to be 8.0 ± 1.0 seconds with a difference of 0.8 ± 0.4 seconds(Shimizu et al, 1974). This has been attributed to the faster blood flow(Perr et al, 1968) and the low peripheral resistance in the choroidal vascular bed (Archer et al, 1970). The early phase of fluorescein angiography is referred to as choroidal flush. The choriocapillaris of the macular region are filled first and thereafter the temporal and nasal regions are filled respectively(Archer et al, 1970, Shimizu et al, 1974). The peripheral

choroid shows a different pattern of angioarchitecture. It is less densly populated with the vessels and the choroidal arteries run close and parallel to the choriocapillaris (Shimizu and Ujii, 1976), and there is also a little difference between the filling of the two system. As soon as the dye reaches the choriocapillaris, it leaks out and give the appearance of a mosaic which is at first discrete and later gets completely filled up. This is known as complete filling stage of choriocapillaris. This is followed by an early emptying phase of choriocapillaris. In this phase the reverse of early filling phase of choriocapillaris occurs. The areas which were empty in the early filling phase are filled up in the late emptying phase and vice versa, forming a honeycomb pattern (Hayreh, 1974). In the midretinal venous phase of dye transit, the dye in the choriocapillaris gives the appearance of a diffuse homogenous background fluorescence which obscures most of the underlying choroidal vessels (Archer et al, 1970). The presence of lighter pigments present less interference with choroidal fluorescence allowing it to be evident earlier in its filling phase (Schatz et al, 1978).

The normal choroidal vascular bed always shows some opacial filling defects varying from 1-2 seconds (Costerhuis and Boen-Tan, 1971), 2 seconds (Hyvarinen et al, 1969) and even upto the early retinal arteriovenous

phase (Archer et al, 1970) and very rarely the filling defects may be observed after the major retinal veins start to fill with fluorescein(Hayreh, 1974). The concept described by Evans et al(1973) and Shimizu et al (1974) that all choroidal filling defects are a physiological phenomenon has not been accepted by Hayreh (1976) who believes that the choroidal filling defects are a definite expression of a pathological process in the choroid.

I-Choroidal Fluorescence in myopia and related disorders

Arm to choroid circulation time has been found to be longer in cases of high myopia (Yoshihara, 1979). The circulation in the choroidal capillary layer of the choroid is greatly obstructed in high progressive myopia. Later on the obliteration and atrophy of the middle sized and large choroidal vessels aggravates the situation. As it is well known that the external retinal layers are supplied with blood from the choroidal capillary layer of the choroid, hence the affect on the choroidal circulation is responsible for the retinal changes in high myopia (Salzhanova, 1978).

The uniform background fluorescence is absent at the sites showing atrophy of choriocapillaris and here the major choroidal vessels are visible throughout all the stages of fluorescein angiography. Normally these vessels are not apparent beyond the early retinal venous

phase of angiography as they are obscured due to the disappearance of the dye from the choriocapillaris (Archer, 1972). The intermediate sized choroidal vessels become apparent when the choriocapillaris are absent (Archer et al, 1970). Pathological defects of larger choroidal vessels along with the loss of choriocapillaris result in total absence of choroidal fluorescence. This is characteristically seen in choroideremia and total choroidal atrophy of degenerative, traumatic or inflammatory aetiology (Archer, 1972). In a group of degenerative and atrophic diseases of the choroidal vessels categorised as choroidal sclerosis, the larger choroidal vessels are seen to be narrower on ophthalmoscopic examination, but are found to be quite patent on fluorescein fundus angiography (Archer et al, 1971). Whenever the pigment epithelium is absent at certain places in myopia, the choroidal fluorescence through the defect is well seen in the early arterial stage during the filling of choriocapillaris. When pigment aggregations are present, they mask the underlying choroidal fluorescence (Rosen , 1969). If the retinal pigment epithelium is absent in the peripapillary area, the circumpapillary fluorescein is also well demonstrated in early phase as choroidal fluorescence and in late phases due to scleral staining (Rosen, 1969). The peripapillary choroidal vasculature is completely seen in about 1/3rd of cases with peripapillary atrophy and in rest of cases

it appears to be incomplete with irregular filling (Stangos et al, 1976). A dense network of choroidal peripapillary framework, formed by the radially arranged choroidal vessels has also been noted in high myopia (Stanogs et al, 1976). They start from the disc and the vessels emerging from points in the periphery of the disc. The peripapillary choroid is partially visible in the crescent area in pathological myopia and has a plexus like appearance. In some cases a second ring of complete or incomplete vascular filling in venous phase has been noticed. It indicates that the peripapillary choroidal circulation consists of a series of concentric circles(Amalric, 1971). The lacquer crack lesions associated with degenerative high myopia do not leak fluorescein as revealed by fluorescein fundus angiography (Watanabe et al, 1976). This shows that lesion is due to healed connective tissue at the ruptured chorioretinal barrier by the elongation of the eye ball.

The ischaemia may also damage the retinal pigment epithelium and the breakdown of the chorioretinal barrier may lead to non-rhegmatogenous serous retinal detachment. In such cases fluorescein fundus angiography reveals the leakage and accumulation of fluorescein dye at the site of lesion, accumulating in the subretinal fluid (Gitter et al, 1968).

3. Macular Region

Fluorescein fundus angiography reveals that the macular region is the meeting point not only of many watershed zones of the various short posterior ciliary arteries but also of all the watershed zones of the vortex veins. This feature helps to understand the frequent and common occurrence of degenerative lesions in the macular area (Hayreh, 1974, 1976).

The macula appears to be dark on fluorescein fundus angiography in normal eyes. This is due to the foveal retinal capillary free zone and the more taller pigment epithelial cells which are rich in high concentrations of xanthophyll pigment and lipofuschin (Schatz et al, 1978) as compared to rest of the retinal pigmentary epithelium.

Fluorescein fundus angiography reveals a number of findings in the macular region of eyes having pathological myopia. Prominent among these are macular haemorrhages without showing any neovascular tissue or leakage of fluorescein dye(Hayashi et al, 1980); with leak and submacular choroidal neovascularization (Levy et al, 1977; Schatz et al, 1978; Spitznas, 1976 b) and with submacular choroidal neovascularization accompanied by proliferation of the pigment(Yoshihara, 1979; Levy et al, 1977). The submacular choroidal neovascularization has to be distinguished from other disorders presenting with the same picture like presumed ocular histoplasmosis

(Gass, 1973; Spitznas, 1976 a), macular drusen and angoid streaks (Hayreh, 1974) and in exudative senile maculopathies (Spitznas, 1976 b). Yoshihara (1979) however, feels that the localised submacular neovascularisation with pigment proliferation is characteristic of only high myopia and no other chorioretinal degeneration. Fluorescein fundus angiography reveals that probably these submacular neovascular tufts in Fuch's spot precede the haemorrhagic or serous detachments (Levy et al, 1977). The presence of a greater pigmentary deposition and associated chorio-retinal atrophy in high myopia is a distinguished feature from the serous and haemorrhagic detachment of the macula in senile non-myopic eye though sub-macular choroidal neovascular tufts have been reported in both the instances (Levy et al, 1977). The leakage of dye in Forster Fuch's spot of high myopia has been some distance from the fovea and fluorescein angiographyis quite helpful when one is contemplating laser treatment (Wessing, 1972; Gass, 1973). Gass (1967) believes that haemorrhagic disciform macular degeneration as seen associated with the Fuch's spot is the result of an acute haemorrhagic detachment followed by organisation of subneuroepithelial haemorrhage and proliferation of the pigment epithelium in a dark spot in the macula. The increased capillary permeability in the choriocapillaris and the breakdown of chorioretinal barrier at the site of fluorescein leak are responsible for the serous

detachment of the retina(Gass, 1967).

One of the important findings in high myopia is the presence of lacquer creck lesion (Klein and Curtin, 1975). On fundus examination these lesions appear as a linear or dendritic figures with faint yellow white colour and on fundus fluorescein angiography, the fluorescence is observed at the site of the lesion just before the arterial phase without any increase in the later phases or leakage of dye (Watanabe et al, 1976; Yoshihara, 1979; Michael Shapiro & Suresh R. Chandra, 1985).

K. Fluorescein fundus angiography of peripheral retina

The watershed zones between the short posterior ciliary arteries and the anterior ciliary arteries located in the peripheral part of the choroid are the second vulnerable part of the choroid and is responsible for most of the degenerative conditions seen in old person and other degenerations in this part (Hayreh, 1974). There are only isolated reports of fluorescein fundus angiography of the peripheral fundus with equatorial degeneration (Wessing and Schlicke, 1976) and rhegmatogenous retinal detachment(Minodo and Kanagami, 1976).

MATERIAL & METHODS

MATERIAL AND METHODS

The present study was undertaken in 38 eyes of 26 individuals at the Department of Ophthalmology, M.L.B. Medical College & Hospital, Jhansi. The subjects were collected from the refraction units of the Department of Ophthalmology, M.L.B. Medical College, & Hospital, Jhansi. The eyes studied were divided into three groups depending upon the history and clinical examination of the fundus.

Group I - (Normal eyes):

This consisted of 6 emmetropic eyes of 4 subjects without any clinical abnormality of the fundus. The media in these eyes was perfectly clear.

Group II - (Simple myopia):

This group consisted of 17 eyes of 12 subjects with a refractive error of -6.0 D spherical to -12.0 D spherical. These were considered to be simple myopic because there were either complete absence or minimal degenerative changes in the central and peripheral fundus although there was a prominent crescent in the majority of the eyes. The macular area was normal and the media were clear.

Group III - (Pathological myopia):

This consisted of 15 eyes of 10 subjects with

a myopia varying from -9.5 D spherical to -24.0 D. spherical. There were wide-spread central and peripheral degenerative changes in the choroid and the retina in these eyes. There was a history of rapid increase of myopia in all the patients. The prominent degenerative changes included a large myopic crescent, peripapillary choroidal atrophy, central macular degenerative changes including Forster Fuch's flecks, extensive chorioretinal atrophy and with or without peripheral retinal degenerations. The ocular media was appreciably clear in majority of the eyes except for in one eye which had vitreous opacities and an early posterior capsular opacity of the lens. However, in these eyes the fundus could be seen and fluorescein angiography was possible.

Every case was examined in detail and the findings were recorded on the proforma(Appendix I).

1. CLINICAL HISTORY

A detailed present, past and family history suggestive of myopia was enquired from each individual.

2. CLINICAL EXAMINATION

(i) Anterior segment

The external examination of each eye was done to exclude any pathology in the cornea, anterior chamber, pupil and lens.

(ii) Slit lamp examination

It was conducted to look for any pathology in the anterior segment and the vitreous. A special attention was paid to study any lenticular or vitreous opacity, vitreous degenerations and liquifaction.

(iii) **Visual acuity and refraction**

Visual acuity of each eye was recorded and in eyes having a poor visual acuity, the pupils were dilated by atropine 1% ointment, homatropine 2% drops or phenylephrine 10% drops depending upon the age of the individual. The retinoscopy under full mydriasis was done and a postmydriatic test was carried out. The refractive error as recorded on postmydriatic test was taken as the refractive error of the individual.

(iv) **Fundus examination**

The central fundus of each eye was studied by the direct ophthalmoscope for the presence of any pathology in the disc, circumpapillary region, blood vessels, centrocaecal area, and the macular area. The status of the choroidal vessels and the retinal pigmentary epithelium was also recorded. The equatorial region of the retina was also visualised by direct

ophthalmoscopy specially in the individuals having high myopia.

3. FLUORESCEIN FUNDUS ANGIOGRAPHY

The fluorescein fundus angiography was undertaken in each patient after a maximal dilatation of the pupils by a suitable mydriatic. Both eyes of 12 subjects were studied in two different sittings. In the remaining 14 subjects only one eye could be studied by fluorescein fundus angiography as the individuals did not turn up for the similar examination of the other eye.

A. APPARATUS

The apparatus used for the present study was Carl-Zeiss fundus camera with automatic control equipment, with high power high speed flash generator meant for fluorescein angiography of the fundus. This instrument basically consists of (1) high power high speed flash generator, (2) automatic camera system, (3) pulse generator, (4) data recording system, (5) filters. The apparatus was used as per manufacturer's instructions. A short account of these parts is given below:

(i) High Power, high speed flash generator

This generator has 4 different steps for flashes of 120 watts/sec; 240 watts/sec; 480 watts/sec; 720 watts/sec. The firing sequence varies from 0.5 seconds at 120 watts/sec - 240 watts/sec to 1.2 seconds to 720

watts/sec. The flash generator mainly consists of a power supply unit with cut outs and selector switches, control unit with filament lamp and a pulse generator.

(ii) Automatic camera system

The automatic camera system is a special purpose Asahi Pentax Camera with a built in tripping magnet driven by an electric motor. This camera is attached to the standard Zeiss fundus camera.

The hinged mirror in the eye piece head is actuated by a rotatory magnet. Automatic photography is initiated by pressing a pedal switch. The camera is ready for the next exposure after a minimum of 0.5 seconds. The power supply unit for the automatic camera system is incorporated in the flash generator. To study the fine details in a small field a 1.5 x auxillary objective can be inserted in the eye piece.

(iii) Pulse Generator

Since it is difficult to take a series of exposures manually at timed intervals of 1-2 seconds, a special automatic timer is attached which triggers the automatic camera system at pre-set intervals as long as the pedal switch is pressed. Automatic timer has three different settings for sequential photography , 0.5 seconds to 3.5 seconds at flash step 60/120/240 watts/ sec. from 0.7 seconds to 3.7 seconds at flash step 420 watts/sec., 1.2 to 4.2 seconds at flash step 720 watts/sec.

(iv) Data Recordings

The study of serial photographs is considerably facilitated by recording the data together with each exposure. The data including the patient's identification number (PID) and exposure time in seconds or minutes is reproduced on the films. This all is done by the Dataphot system attached to the back of the Camera.

(v) Filters and Films

The selection of a proper combination of exciter and barrier filters is extremely important for the best results. Satisfactory results will only be obtained if the different spectrum distribution factors like an emission flash tube, transmittance of the exciter and barrier filters, activation spectrum, fluorescence spectrum of the fluorescein in the blood plasma, reflectivity of the retina, and film speed are favourably balanced. The exciter filter isideal which removes all the wavelengths from the incident light except in the range of 490-510 nm wavelengths which is the absorption peak of fluorescein excitation. An ideal barrier filter should cut off all the wavelengths except those in the range of 500-520 nm which is the fluorescent or emitted peak of fluorescein. The suitable flash intensity and the proper use of films is also necessary to have good results.

In the present study the filters used with the apparatus were LP 520 barrier filter inserted in the funnel stop and held in front of the camera and a Med 485 excitor filter. The excitor filter is present in the diaphragm turret of the fundus camera. The funnel stop containing the barrier filter can be introduced in the camera body by a simple manipulation. ORWO, 400 ASA, 35 mm film was used in the present investigation. Pictures were taken with the above filter combinations utilising a flash step at 240 watts/sec. Unfortunatily during the period of this study the automatic motarised film advancement system and data phot system were not working properly, so the series of photographs were taken by manual film advancement by quick action cocking lever & with the release of auxiliary & sutter, by auxiliary release Knob and Sutter release knob respectively.

B. PRIPARATION OF THE PATIENT AND TECHNIQUE

Before subjecting the patient for actual procedure the patient was prepared psychologically and mentally for the procedure. After that a fluorescein sensitivity test was done in every individual by administering subcutaneous 0.1 ml to 0.2 ml fluorescein dye prior to the start of the actual procedure. In the present study, only two individuals got mild hyper-sensitivity reactions in the form of nausea, vomiting and giddiness and consequently the procedure was abandoned. They were treated by intravenous steroids

& antihistamines.

The procedure was undertaken in patients, in which no sensitivity to fluoresceinwas observed. The patient was comfortably seated over the stool and the proper focussing of the central fundus was done. The apparatus was made ready for operation after all the necessary and finer adjustments.

After, the instrument was ready for operation, 3 ml of 20% fluorescein sodium dye was injected intravenously in the anticubital vein in majority of the patients and in the veins of the dorsum of the hands in patients of fatty constitution. The dye was injected rapidly within 3-5 seconds with a 19 bore needle.

The time in the wrist watch was noted. The fundus photographs were taken after swinging in the exciter filter knob before the dye injection was commenced. A series of photographs were taken approximately at the interval of 1-2 seconds upto 20 seconds and thereafter the late exposures were taken approximately at 1 minute, 3 minutes, 5 minutes, 10 minutes and 15 minutes intervals with the help of wrist watch. The focussing of the fundus image was frequently checked in between the exposures as patients are often inco-operative or blink the eyes consequent to high speed flash.

The whole procedure lasted for about 15 minutes and in the end the patient was asked to take rest in a supine pisition. No case in the present study developed complications after the procedure was over. Although a likelihood of yellowish discolouration of urine was explained in each individual.

After the completion of the procedure the apparatus was switched off and the film was sent for processing immediately. The universal developer (IFC - 163 developer Powder) was used for the processing. The concentrated stock solution was made as per the manufacturer's instructions. The film was processed in solution consisting of one part of concentrated stock solution and three parts of water in a dark for 4-8 minutes at 25° - 30° temperature. Hyposolution having metabisulphite was used for fixing it and finally the film was washed with water.

C. INTERPRITATION OF FLUORO-ANGIOGRAMS

The fluorescein fundus angiograms were studied in the following way:

(i) Diameter of the disc

The diameter of the disc was measured from prints made from the angiograms at a fixed enlargement in each eye. The figures so obtained for horizontal and vertical diameters of each eye in all the three groups

were compared.

(ii) Calibre of the retinal vessel

The calibre of the retinal vesseles near the disc margin was measured, by projecting the film at fixed distance under same magnification. Absolute values were not obtained, just comparission was done in all the three groups.

(iii) Calibre of choroidal vessels

The measurement of the large sized choroidal vessels was calculated in eyes having pathological myopia by projecting the film at a fixed distance. It was not possible in normal emmetropic eyes or eyes with simple myopia because of the presence of a diffuse and mottled choroidal fluorescence which obscured the underlying choroidal vessels. The choroidal vessels could be measured in eyes with pathological myopia (Group III). The absolute values were not obtained and just a comparative study in the eyes with low refractive error and the eyes with high refractive error in the same group (Group III) was made. The choroidal vessels on the nasal side of the disc were taken into consideration for the comparative study.

(iv) Choroidal fluorescence

The circumpapillary choroidal fluorescence and the fluorescence on the nasal and temporal part of

the choroid in both early and late phases of dye transit were observed and compared. A special attention was paid to the presence of visible choroidal vessels, peripapillary choroidal plexus, chorioretinal anastomosis and the presence of choroidal sclerosis. The early and late fluoreangiograms of the eyes having peripapillary or juxtapapillary choroidal atrophy were also observed to see the nature of fluorescence in the atrophic areas.

(v) Disc fluorescence

The fluorescence of the disc was compared between early and late angiograms. The nature and location of the crescent was similarly studied in both the early and late phases of the dye transit.

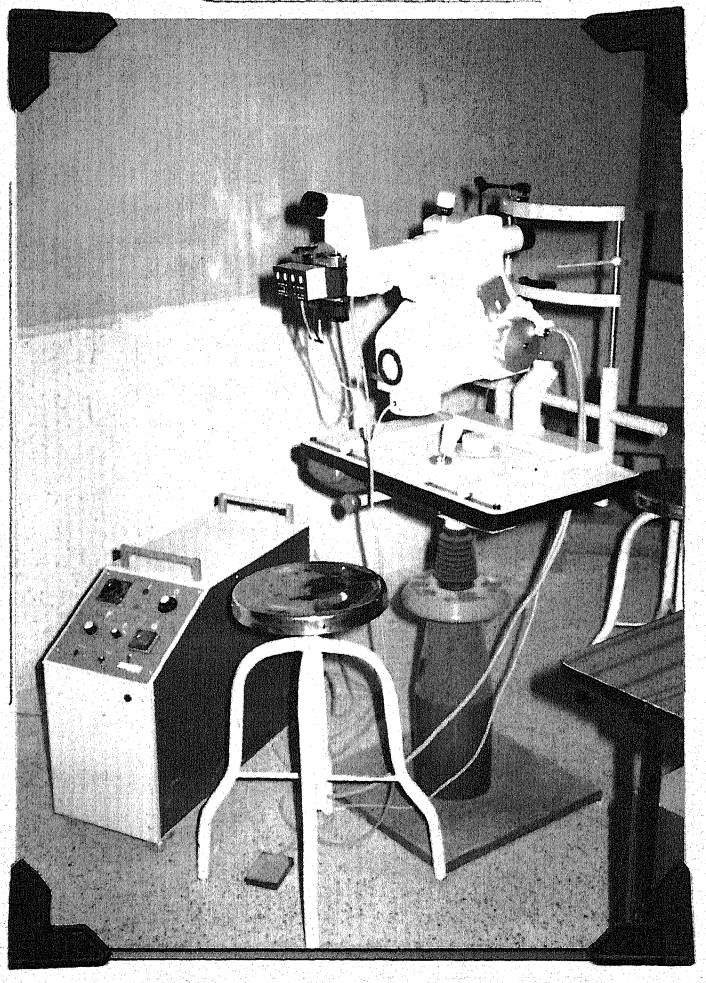
(vi) Retinal fluorescence

Various phases of the dye transit namely the early arterial, arteriovenous, early and late venous phases were studied from the fluorangiograms.

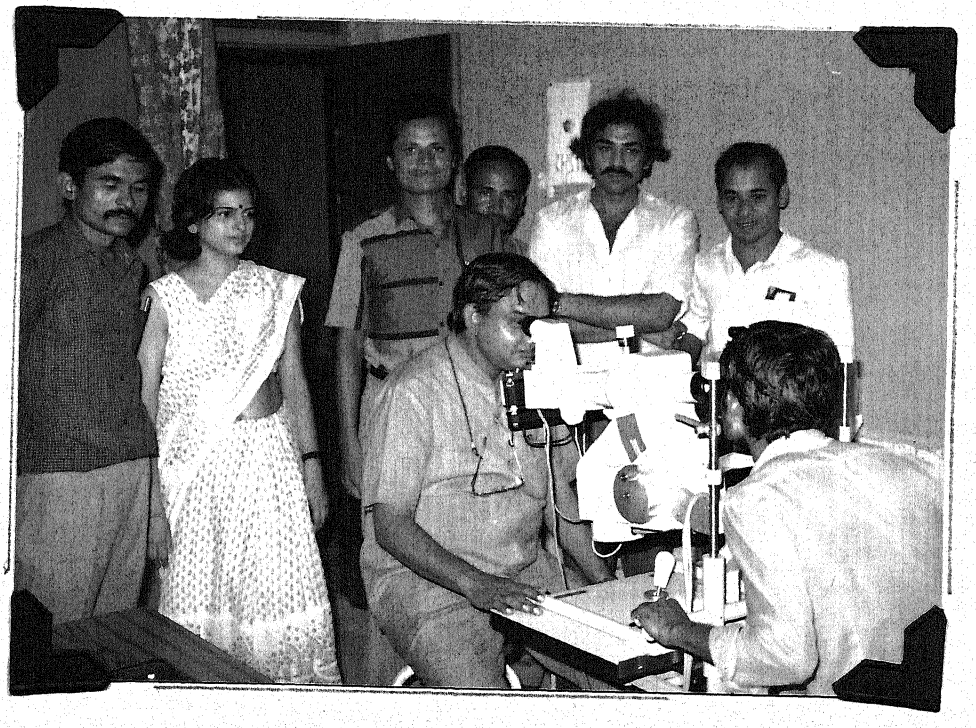
(vii) Macular region

The macular area was observed for the presence of diffuse or localized hyperfluorescence, choroidal atrophy, choroidal sclerosis and subretinal neovascular membrane.

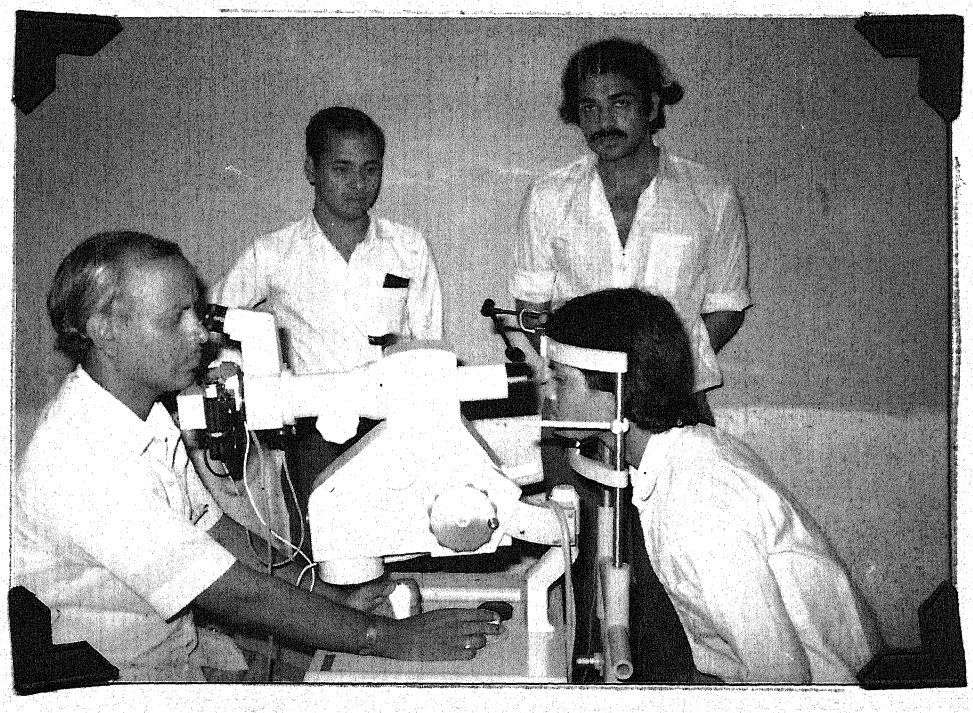
The findings thus obtained in each group were finally compared in all the three groups.



CARL ZEISS FUNDUS CAMERA WITH AUTOMATIC CONTROL SYSTEM, HIGH POWER HIGH SPEED FLASH GENERATOR & PULSE GENERATOR.



DR. G. D. GUPTA,
Head, Department of Ophthalmology.
DEMONSTRATING FUNDUS FLUORESCEIN ANGIOGRAPHY.



DR. B. S. JAIN

Lecturer, DEPARTMENT OF OPHTHALMOLOGY.

DOING FUNDUS FLUORECEIN ANGIOGRAPHY.

OBSERVATIONS

OBSERVATIONS

The observations are based on investigations undertaken in 38 eyes of 26 individuals. They have been divided into three groups (Table I).

Group I - Normal eyes.

Group II - Eyes with simple myopia.

Group III - Eyes with pathological myopia.

A. GENERAL OBSERVATIONS

(i) Age and Sex:

The individuals studied in the present investigation ranged between 13 and 52 years (Table II). The patients affected with simple myopia (Group II - 12 cases) ranged from 15 years to 48 years. The maximum number of patients were in the third decade of life (50.0%). The patients affected with pathological myopia (Group III - 10 cases) ranged from 13 years to 52 years. The maximum number of affected individuals belonged to third decade of life (40.0%). Out of 26 subjects studied, 16 were males and 10 females (Table I).

(ii) Heredity (Table I):

In group II (simple myopia) a positive family history of myopia was noted in 7(58.33%) out of 12 affected individuals. In group III (Pathological myopia) a positive family history of myopia was observed in 8(80.0%) out of 10 affected patients. Overall family history of myopia in both

TABLE - I : DISTRIBUTION OF CASES WITH SEX AND HEREDITY.

Patients sex, Heredity and Eyes.	Group I (Normal)	Group II (Simple myopia)	Group III (Patholo-gical myopia)	Total
1. Number of patients(cases)	4	12	10	26
2. Male	3	7	6	16
Female	1	5	4	10
3. Positive history of myopia.	-	7	8	15
4. Number of Eyes.	6	17	15	38

TABLE- XI : NUMBER OF CASES IN RELATION TO AGE IN GROUP I (NORMAL), GROUP II (SIMPLE MYOPIA) AND GROUP III (PATHOLOGICAL MYOPIA).

Age groups in years	GROUP I		GROUP II		GROUP III	
	No. of Patients	Percen-tage	No. of cases.	Perce-ntage	No. of cases.	Perce-ntage
11 - 20*	2	50.00	4	33.33	2	20.00
21 - 30	2	50.00	6	50.00	4	40.00
31 - 40	-	-	1	8.33	2	20.00
41 - 50	-	-	1	8.33	1	10.00
51-60**	-	-	-	-	1	10.00
Total	4	100.00	12	100.00	10	100.00

*Minimum age of the patient was 13 years.

**Maximum age of the patient was 52 years.

groups II and III was observed in 15 out of 26 cases (57.7%).

(iii) Refractive error (Table III):

The eyes affected with simple myopia (Group II) ranged from -6.0 D spherical to -13.0 D spherical. The maximum number of eyes were between -6.0 D spherical to -9.0 D spherical (14 eyes -82.2%). The eyes affected with pathological myopia (Group III) ranged from -9.5 D spherical to -24.0 D spherical. Out of these, the maximum number of eyes affected were between -12.0 D spherical to -20.0 D spherical (12 eyes -80.0%).

(iv) Visual acuity (Table IV):

11(64.7%) out of 17 eyes (Group II) had corrected visual acuity of 6/6 to 6/9 . The remaining 6 eyes also had substantially good visual acuity (upto 6/24). The eyes affected with pathological myopia had invariably a poor visual acuity because of central degenerative changes. The decrease of visual acuity was more or less proportionate with the increase of myopic refractive error and macular lesions. The corrected visual acuity in this group ranged between 3/60 to 6/24.

(v) Clinical examination:

The external appearance of the myopic eye was that of a large prominent eye with a moderately deep anterior chamber. One individual had a divergent strabismus and one

TABLE - III : DISTRIBUTION OF CASES IN RELATION TO REFRACTIVE ERROR IN GROUP II AND GROUP III.

Degree of Myopia	Group II		Group III	
	No. of eyes	Percentage	No. of eyes	Percentage
*1. -6.0 D Sph. to -7.0 D Sph.	9	52.94	-	-
2. -7.25 D. Sph. to -9.0 D Sph.	5	29.41	-	-
**3. -9.25 D Sph. to -11.0 D Sph.	2	11.76	1	6.66
4. -11.25 D Sph. to -13.0 D Sph.	1	5.88	4	26.66
5. -13.25 D Sph. to -16.0 D Sph.	-	-	5	33.33
6. -16.25 D Sph. to -20.0 D Sph.	-	-	3	20.00
7. -20.25 D Sph. to -24.0 D Sph.	-	-	2	13.33
	17	100.00	15	100.00

*Minimum refractive error in simple myopia -6.0 D Sph.

**Minimum refractive error in pathological myopia -9.5 D Sph.

TABLE - IV : SHOWING RELATIONSHIP OF CORRECTED VISUAL ACUITY IN EYES WITH SIMPLE MYOPIA (GROUP II) AND PATHOLOGICAL MYOPIA (GROUP III).

Corrected visual acuity	Group II		Group III	
	No. of eyes	Percentage	No. of eyes	Percentage
1. Upto 6/60	-	-	4	26.66
2. 6/36 to 6/24	2	11.76	11	73.33
3. 6/18 to 6/12	4	23.52	-	-
4. 6/9 to 6/6	11	64.70	-	-
Total	17	100.00	15	100.00

case had an anisometropic amblyopia. One eye showed vitreous opacities with a complicated cataract but the fundus was seen and fluorescein angiography was possible.

B. OPHTHALMOSCOPIC EXAMINATION

(a) General fundus:

All the 6 eyes in group I had a normal fundus showing no pathology. The eyes with simple myopia (Group II) had tigroid fundii with minimal chorioretinal degenerations. In eyes with pathological myopia (Group III), a number of ophthalmoscopic findings were observed clinically both in the central and peripheral fundus. Extensive chorioretinal degenerations with choroidal sclerosis were observed in all these eyes. One eye showed a dense pigmented clump superotemporally and 1(6.7%) eye showed a localised pigmentary epithelium defect on the infero nasal quadrant. Widespread pigment dispersion of variable density was also noticed at places in the fundi of all the eyes (Group III) more so around the disc.

(b) Disc:

All the 6 eyes (Group I) had a normal disc. The temporal pallor of the disc was observed in 10(58.2%) eyes in group II and in all the eyes in group III. The size of the optic disc was larger in the myopic eyes than the normal eyes and no cupping was noticed in any eye.

(c) Disc Crescent (Table V):

In 13(76.5%) out of 17 eyes (Group II) and in 10(66.7%) out of 15 eyes (Group III), the crescent of various sizes and in various locations around the disc was observed. This is clear from the table that in 11(64.7%) eyes (Group II) and 7(46.6%) eyes (Group III), the temporal crescent was the commonest type observed either alone or involving the superior and inferior margins of the disc. In group II and III, diffused pigment dispersion of various density was also observed at places in the crescent area.

(d) Peripapillary region:

This region showed no pathology in both the group I and II. In group III, this region showed circumpapillary atrophy in 5(33.3%) eyes with diffused pigment despersion in the atrophic area as elsewhere in the fundus.

(e) Blood vessels:

Retinal vessels showed no abnormality in all the normal eyes (Group I). Nasal obliquity of the retinal vessels was observed in 11(64.7%) out of 17 eyes (Group II) and in 9(60.0%) out of 15 eyes (Group III). The retinal vessels appeared to be attenuated to some extent in group II and group III as compared to normal eyes.

(f) Macula:

In all the 6 eyes (Group I) and 17 eyes (Group II), macula showed no pathology and the foveal reflex was intact.

In group III (pathological myopia) various clinical findings were observed in the macular and paramacular area. Dull foveal reflex with pigment mottling was observed in 4(26.7%) eyes. An irregular darkish black patch (Forster Fuch's Flecks) was observed in the macular area(2 eyes) and paramacular area (1 eye). In 1 out of these 3 eyes, two small sized pigmentary epithelium defects were also observed on the sides of the Forester Fuch's flecks. Widespread chorioretinal atrophic patches (1 eye -6.7%) and choroidal sclerosis (1 eye -6.7%) were also observed surrounding the macular area.

Peripheral Fundus:

Cystoid degeneration in 3(20.0%) and lattice degeneration in 1(6.7%) eyes respectively were noticed in group III.

C. FLUORESCEIN FUNDUS ANGIOGRAPHY

The observations of fluorescein fundus angiography in all the three group (Group I, II and III) are described collectively under the following headings.

(1) Choroidal Fluorescence:

In 4(66.7%) out of 6 eyes (Group I) a uniform choroidal fluorescence was observed soon after the injection of the dye. In 1(16.6%) eye, an early marked choroidal fluorescence was seen in the superonasal quadrant, while in the remaining 1 eye, this was visible on the nasal and

TABLE - V : TYPES OF DISC CRESCENT IN EYES WITH SIMPLE AND PATHOLOGICAL MYOPIA.

Type of Crescent	Group II		Group III	
	No. of eyes.	Percentage	No. of eyes.	Percentage
1. Temporal	9	52.94	3	20.00
2. Superior and Inferior temporal	2	11.76	4	26.66
3. Inferior temporal	1	5.88	-	-
4. Inferonasal	-	-	-	-
5. Annular	1	5.88	3	20.00
6. Indistinct (Merged with peripapillary atrophy)	-	-	5	33.33
Total	13	76.47	10	66.66

inferotemporal quadrant. However, these two eyes also revealed a uniform choroidal fluorescence in the arterio-venous phases.

In 12(70.5%) out of 17 eyes (Group II), a uniform choroidal fluorescence was observed soon after the injection of the dye. Interestingly in 5(29.5%) eyes, the choroidal filling was observed to start initially from the temporal quadrants, specially the superotemporal quardrant. Later with the passage of dye the entire choroidal bed showed a uniform fluorescence both on the nasal and temporal sides of the disc.

In group III (pathological myopia), 11(73.3%) eyes showed a characteristic nasal choroidal fluorescence. It was more prominent in 4(26.7%) eyes and no temporal choroidal fluorescence was observed even in the late venous phases, 4 eyes, where such a feature was present, had a refractive error ranging from -14.0 D spherical to -24.0 D spherical.

In 6(40.0%) out of above 11 eyes, the nasal choroidal fluorescence was comparatively more prominent than the temporal choroidal fluorescence.

In the remaining 4(26.7%) eyes, a uniform choroidal fluorescence was observed on nasal and temporal sides of the disc, which persisted even in the late venous phases. These eyes, where a uniform choroidal fluorescence was observed, had a refractive error ranging from -9.5 D

spherical to -16.0 D spherical.

In group III, the individual choroidal vessels could be observed in 11(73.3%) eyes. Of these 6(40.0%) eyes showed a uniform filling of choroidal vessels and no empty vessel was observed. In the remaining 4(26.7%) eyes, although the choroidal vessels could be seen this feature was not uniform so much so that a few of the individual choroidal vessels could be seen free from fluorescein in early and late fluoroangiograms. These 11 eyes had a refractive error ranging from 11.25 D spherical to -24.0 D spherical.

(2) Choroidal vessel calibre:

Fluorescein angiography did not reveal any individual choroidal vessels in groups I and II. However, in group III, individual choroidal vessels could be observed in 11(73.3%) eyes, and thus the choroidal vessel calibre could be measured in these eyes. The average calibre of large sized choroidal vessels was found to be approximately 1 mm (8 times magnification of the negative) in 5(33.3%) eyes with a refractive error ranging from -9.5 D spherical to -16.0 D spherical and 0.5 mm (8 times magnification of the negative) in remaining 6(40.0%) eyes with a refractive error of -18.0 D spherical to -24.0 D spherical.

(3) Disc Fluorescence:

A uniform and generalised filling of the disc

was seen in all the three groups. In the early arterial phase, the disc was seen to fill from the temporal side in all the three groups. The filling gradually increased as the dye passed from the arterial to the venous phases and finally the whole disc showed a complete fluorescence.

(4) Diameter of the disc:

The average horizontal disc diameter group I, II & III was found to be 1.6 mms ± 0.4 mms; 2.0 mms ± 0.4 mms; and 2.4 mms ± 0.4 mms respectively, while the average vertical disc diameter was found to be 2.0 mms ± 0.4 mms; 2.8 mms ± 0.4 mms; 2.4 mms ± 0.4 mms respectively.

(5) Disc crescent:

The crescent was not seen in the normal eyes (group I). Different types of disc crescent were seen in 13(76.47%) out of 17 eyes (Group II) and in 10(66.67%) out of 15 eyes (Group III). The fluoresangiographic findings were similar to the ophthalmoscopic findings. In the early arterial phase only the extreme temporal edge ofthhe crescent revealed slight fluorescein which gradually increased with the passage of the dye. In the late phases, the staining of the crescent of variable density, was invariably observed in all the eyes so much so that it became difficult to distinguish it from the disc border. The choroidal vessels from the peripapillary choroidal plexus or circle of Zinn underlying the crescent area were observed in 4(23.5%) eyes in group II and in

5(33.4%) eyes in group III. These eyes also revealed the chorioretinal anastomosis in the crescent area.

(6) Peripapillary region:

The peripapillary region revealed no pathology in group I and group II except for the appearance of choroidal vessels from the peripapillary plexus underlying the crescent area in group II.

The peripapillary region in eyes with pathological myopia (Group III), revealed the following characteristic patterns.

(i). Peripapillary choroidal plexus:

In 5(33.4%) eyes, localised areas of peripapillary choroidal plexus were observed adjoining the temporal crescent, although the individual choroidal vessels could not be seen. In 3(20.0%) eyes, the peripapillary choroidal plexus was observed in the area of circumpapillary atrophy. In the remaining eyes, this plexus could not be identified and only a patchy hyper fluorescence in the region of peripapillary area was observed.

(ii). Peripapillary choroidal atrophy:

Peripapillary atrophy was observed in 6(40.0%) eyes and was seen in two forms.

(a) Circumpapillary choroidal atrophy (5 eyes):

Fluorescein angiography in these eyes revealed

an irregular underlying choroidal vascular pattern in the early arterial phases. In 2(13.3%) out of these 5 eyes a diffused hyperfluorescence in the area of atrophy, starting initially from the margins and gradually involving the entire area of the circumpapillary atrophy, though not in a uniform pattern was observed. In 3(20.0%) out of above 5 eyes, however, the diffuse hyperfluorescence in the area of circumpapillary choroidal atrophy was not observed at all even in the late venous phases and only a minimal marginal fluorescence was observed. The refractive error of the eyes (2 eyes) showing a late diffused hyperfluorescence in the area of circumpapillary choroidal atrophy ranged between -9.5 D spherical to -18.0 D spherical, while the refractive error of the eyes (3 eyes) not revealing this feature was -24.0 D spherical.

(b). Juxtapapillary or localised atrophy (1 eye):

In this eye, having a refractive error of -20.0 D spherical, the choroidal atrophy was observed in the form of an irregular localised area adjacent to the inferonasal border of the disc, which did not reveal hyperfluorescence even in the late venous phases.

(7) Cilioretinal artery:

The cilioretinal artery was observed in 2(33.3%) eyes in group I and only in 1(5.8%) eye in group II. As the dye passed through the early arterial phase, the cilioretinal artery was seen tobe well delineated, showing a uniform

fluorescence throughout its course. In 1 eye (Group I), two cilioretinal arteries, one above and one below, were seen to arise from the temporal border of the disc and extended up to the parafoveal area. In another eye(Group I) it was seen to arise from the temporal border of the disc in a hooked manner and soon divided into two branches just out side the disc border. The branches were observed to extend beyond the foveal region and gave fine twigs(macular capillaries) to the macular region.

In the eye belonging to group II, the cilioretinal artery was also seen to arise in a hooked manner from the superotemporal border of the disc and was observed to extend towards the macular region, giving smaller twings to this area.

(8) Arterial phase:

Soon after the dye reached the central retinal artery, the whole arterial tree was fluorescent in all the three groups.

(9) Capillary phase:

The dye reached the capillaries from the arteries and precapillary arterioles in a quick succession and only two type of capillaries could be seen in all the three groups.

(i). Papillary and peripapillary capillaries:

These were seen to arise from the retinal vessels

in the disc region, extending radially towards the disc margin, the crescent area and upto the peripapillary region. In 4(23.5%) out of 17 eyes (Group II) and 5(33.3%) out of 15 eyes (Group III), chorioretinal anastomosis were also observed.

(ii). Macular capillaries:

Capillaries in the macular area consist of the concentric vascular loops surrounding a central avascular area. Individuals vessels could be identified in the parafoveal region in all the three groups. These were poorly seen on ophthalmoscopic examination. The cilioretinal artery also contributed to these capillaries.

(10) Arteriovenous phase:

In all the three groups, the smaller venous radicles at the macula fill first. The larger veins show fluorescence only along the vessel wall initially and subsequently the whole vein is filled up completely with the dye. The veins appear to be prominent and wider.

(11) Late venous phase (Arteriovenous recirculation phase):

In the later stages, with the recirculation of the dye, the fluorescence of decreased intensity was observed in the retinal vessels till the whole of the dye was washed off the circulation in some 10-15 minutes after the injection of dye. No pathology was observed in the retinal

vessels during this phase of dye transit in all the three groups.

(12) Calibre of the retinal vessels:

The average calibre of big branches of the central retinal artery, measured at the disc margin was found to be in the ratio of 1 : 0.73 : 0.67 in group I, II and III respectively. The calibre of the big tributaries of the central retinal vein measured at the same site was found to be in the ratio of 1: 0.69 : 0.68 in group I, II and III respectively.

(13) Macular region:

The macular area in both groups I and II appear obscuring the underlying choroidal fluorescence. No macular pathology was seen in both the groups I and II.

In group III, a number of fluorangiographic findings were observed in macular and paramacular region in 11(73.3%) eyes out of 15 eyes (Table VI). It is evident that the localized or diffused macular or paramacular hyperfluorescence was the commonest finding. While localized or patchy hyperfluorescence was observed in the paramacular area of 1 eye and diffuse hyperfluorescence was observed in another one eye.

In 3(20.0%) eyes, where a darkish black patch (Forster Fuch's flecks) was observed in the macular (2 eyes) and paramacular area (1 eye) on ophthalmoscopic examination, fluorangiographic pattern was characteristic.

TABLE - VI : FLUORESCEIN FUNDUS ANGIOGRAPHIC FINDINGS SHOWING MACULAR AND PARAMACULAR LESIONS IN EYES HAVING PATHOLOGICAL MYOPIA (GROUP III)

Fluorangiographic findings	No. of eyes	Percentage
1. Macular or paramacular hyperfluorescence	4	26.7
2. Localised Hyperfluorescence	1	6.7
3. Diffused Hyperfluorescence	1	6.7
4. Hyperfluorescence at the site of Forster Fuch's flecks	3	20.0
5. Central choroidal atrophy	1	6.7
6. Central choroidal sclerosis	1	6.7
Total	11	73.3

In 4 eyes macular degeneration was detected Ophthalmoscopically but no any abnormality detected fluorangiographically.

In 2(13.3%) eyes out of these above 3 eyes a hyperfluorescent zone was surrounded by a nonfluorescent zone of variable density. In 2(13.3%) eyes out of above 3 eyes, no associated pigmentary epithelium defects were seen on ophthalmoscopy and fluorescein angiography revealed a dense hyperfluorescent area in 1(6.7%) eye and a less marked diffuse paramacular hyperfluorescence in one eye. In the thrid eye showing Forster Fuch's Flecks , two hyperfluorescent patches meeting in the centre lying close to each other were seen in the late venous phases. These two hyperfluorescent corresponds to the pigmentary epithelium defects at the same places except that these areas appeared smaller and separate on ophthalmoscopic examination, and Forster Fuch's Flecks, was seen in between these two defects.

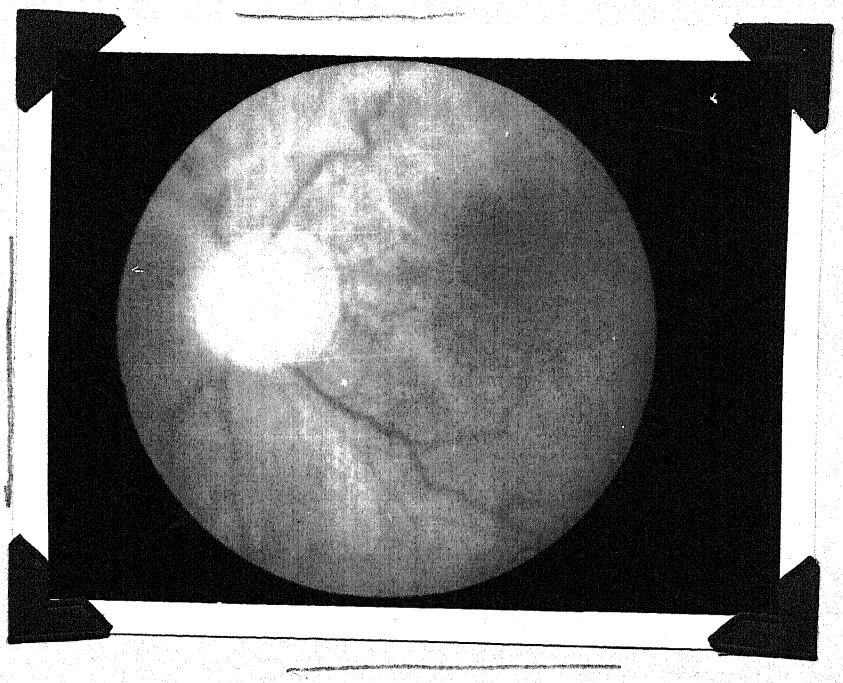
In another 2(13.3%) eyes the macular and paramacular area revealed sclerosed choroidal vessels and choroidal atrophy respectively.

In the remaining 4(26.7%) eyes although pigment mottling and dull foveal reflex were observed on ophthalmoscopi examination, fluorescein fundus angiography, however, did not reveal any pathology.

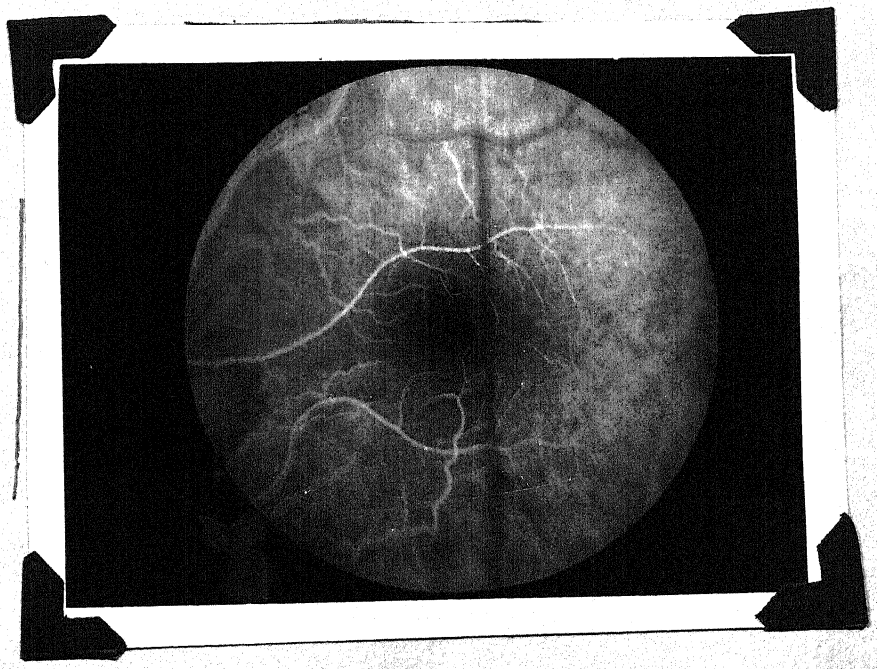
(i) Miscellaneous:

In eyes with simple myopia (Group II), fluorescein fundus angiography revealed no pathology, although the fundii were tessalated in almost all the eyes. In 1(6.7%) eye with

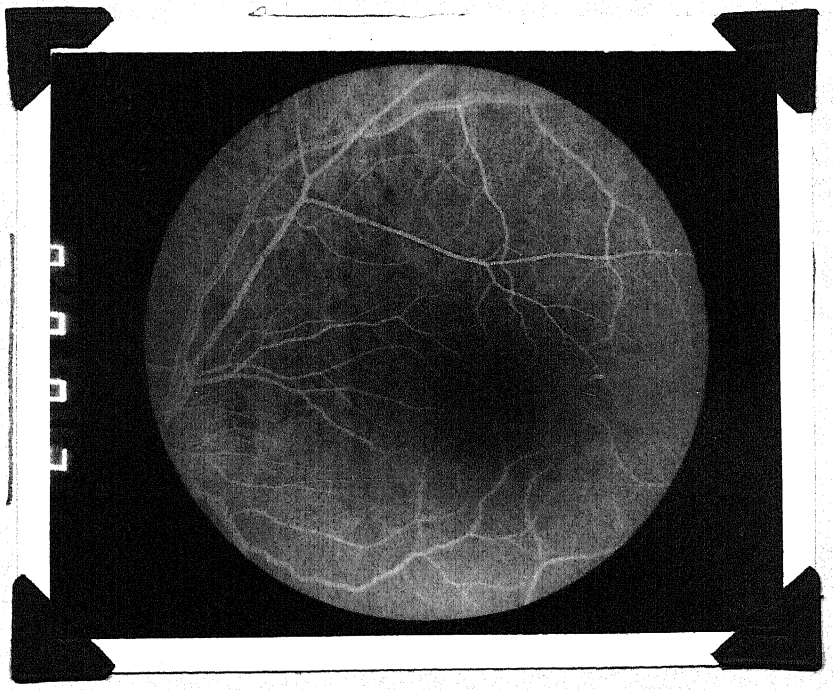
pathological myopia (Group III), fluorescein fundus angiography revealed a localised hyperfluorescent area on the inferonasal quadrant near the disc in the region of ophthalmoscopically detectable pigmentary epithelium defect. In 2(13.3%) eye, fluorescein angiography revealed a nonfluorescent patch at the site of ophthalmoscopically detectable large pigmentary clump on the superotemporal quadrant.



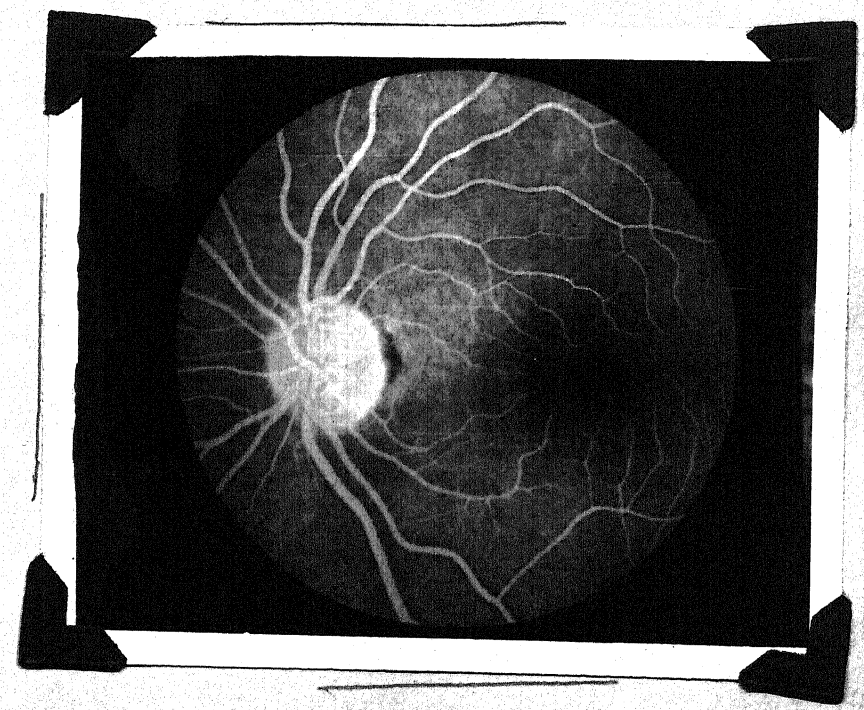
ANGIOGRAM SHOWING VERY EARLY PHASE OF DYE TRANSIT, ONLY CHOROIDAL FLUSH & DISC FLUORESCENCE CAN BE SEEN, THERE IS NO DYE IN THE RETINAL VESSELES.



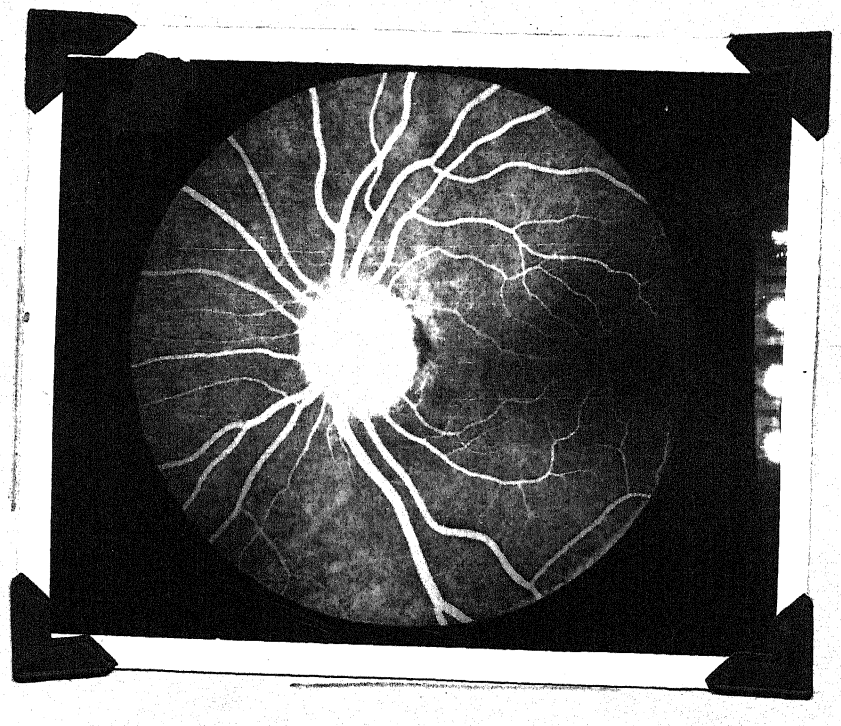
ANGIOGRAM SHOWING LATE ARTERIAL PHASE OF DYE TRANSIT, FILLING OF THE VENOUS RADICLES AT THE MACULA STARTED, AND UNIFORM CHOROIDAL FLUORESCENCE CAN BE SEEN.



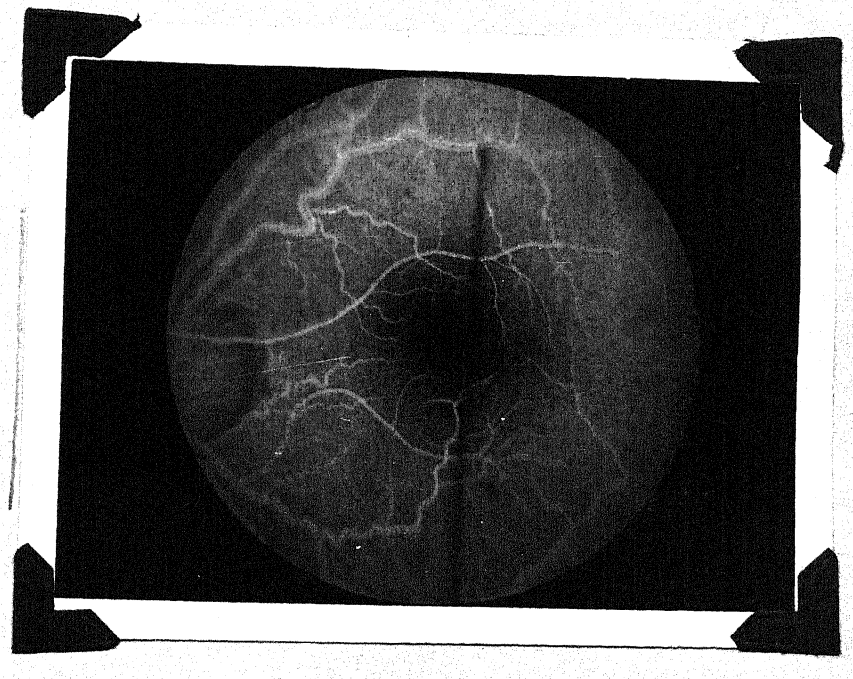
ANGIOGRAM SHOWING EARLY ARTERIO-VENOUS PHASE OF THE DYE, LAMINAR PATTERN OF VENOUS FLOW IN LARGER VEINS, UNIFORM CHOROIDAL FLUORESCENCE & CAPILLARY NET WORK OVER THE DISC.



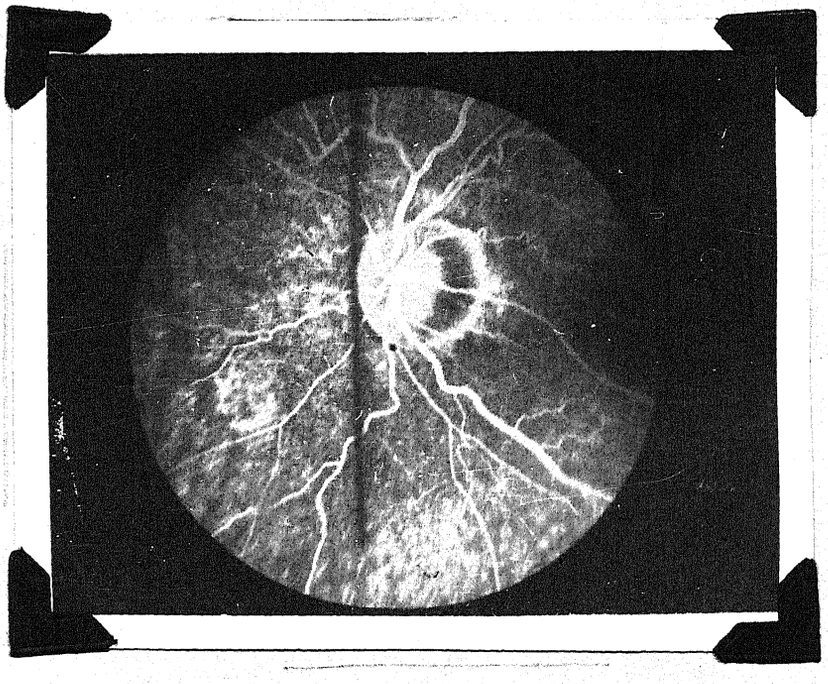
FLUORESCEIN ANGIOGRAM REVEALING DOMINANT CHOROIDAL FLUORESCENCE IN ARTERIOVENOUS PHASE OF THE DYE TRANSIT AT THE SUPERIO-TEMPORAL QUDRANT, AND COMPLETE DISC FLUORESCENCE.



ANGIOGRAM SHOWING COMPLETE FLUORESCENCE OF DISC, SOME WHAT ENLARGED AVASCULAR MACULAR ZONE AND STILL UNSTAINED CRESCENT AREA, IN LATE ARTERIO-VENOUS PHASE.

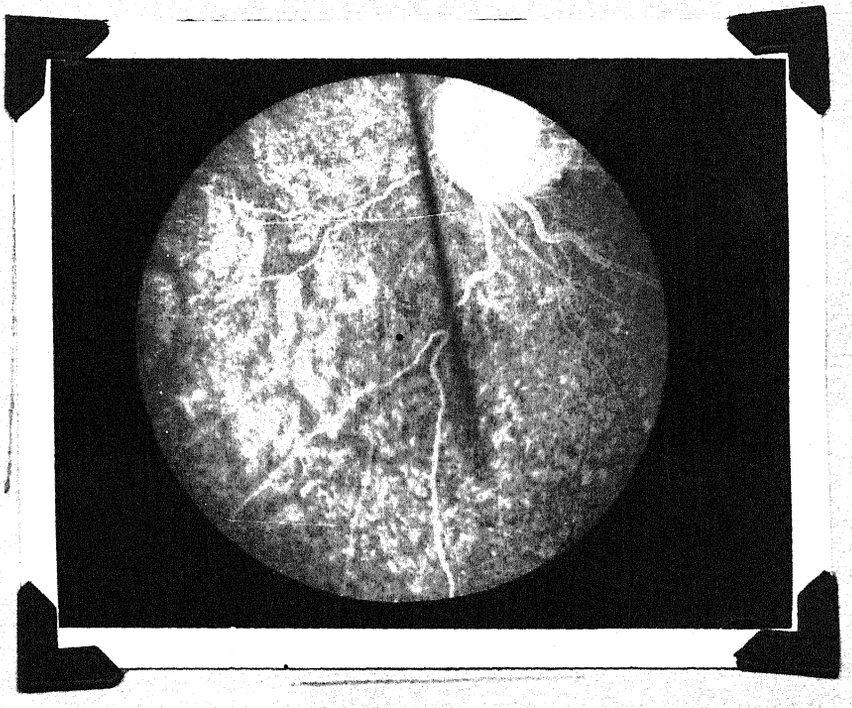


FLUORESCEIN ANGIOGRAM SHOWING EARLY VENOUS PHASE, UNSTAINED TEMPORAL CRESCENT, FILLED VENOUS RADICLES AT THE MACULA & DARK AVASCULAR MACULA.



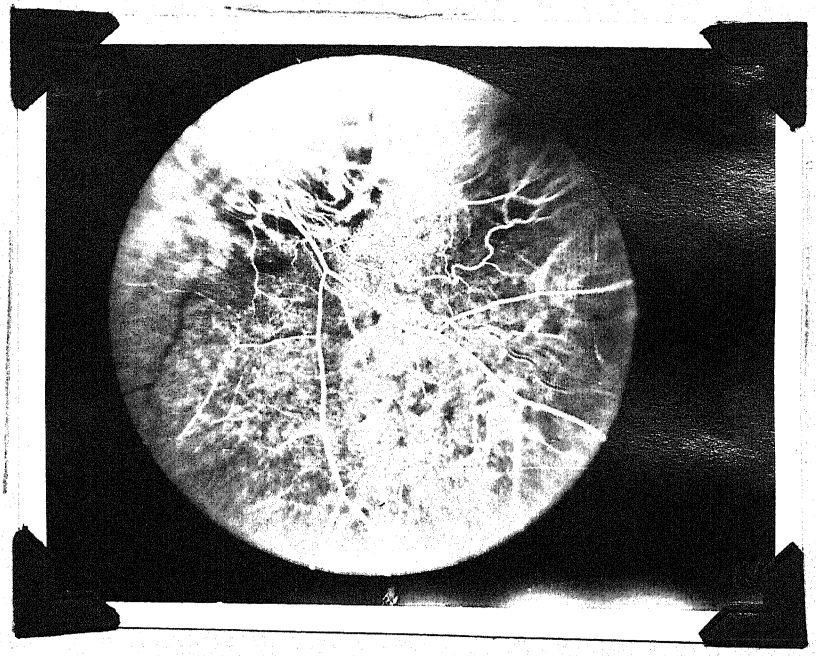
EARLY ARTERIOVENOUS PHASE

- UNSTAINED CLEARLY DISTINCT TEMPORAL CRESCENT.
- NON UNIFORM CHOROIDAL FLUORESCENCE.
- LESS DISTINCT CAPILLARY NET WORK.

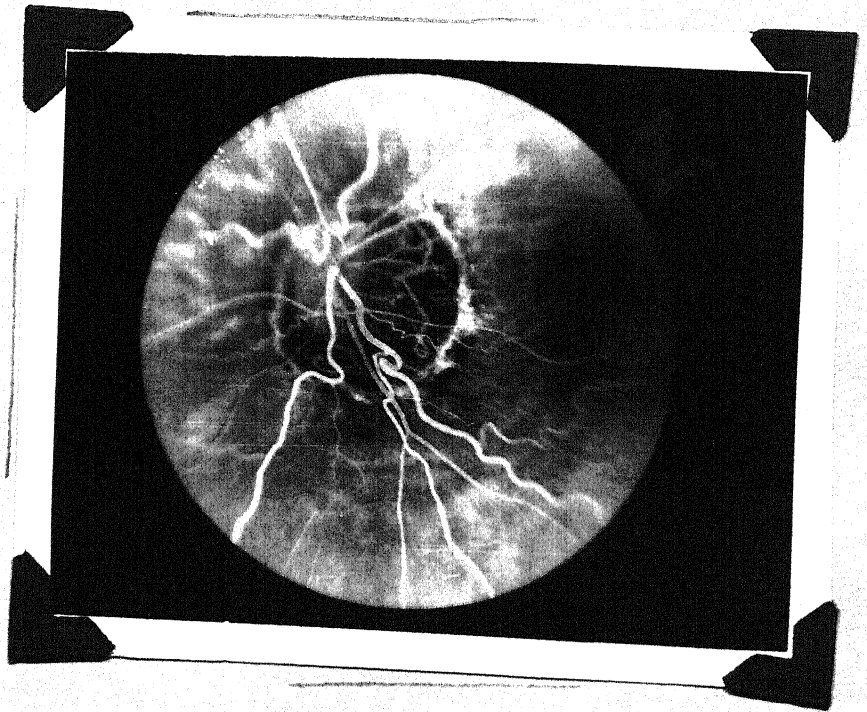


LATE ARTERIOVENOUS PHASE.

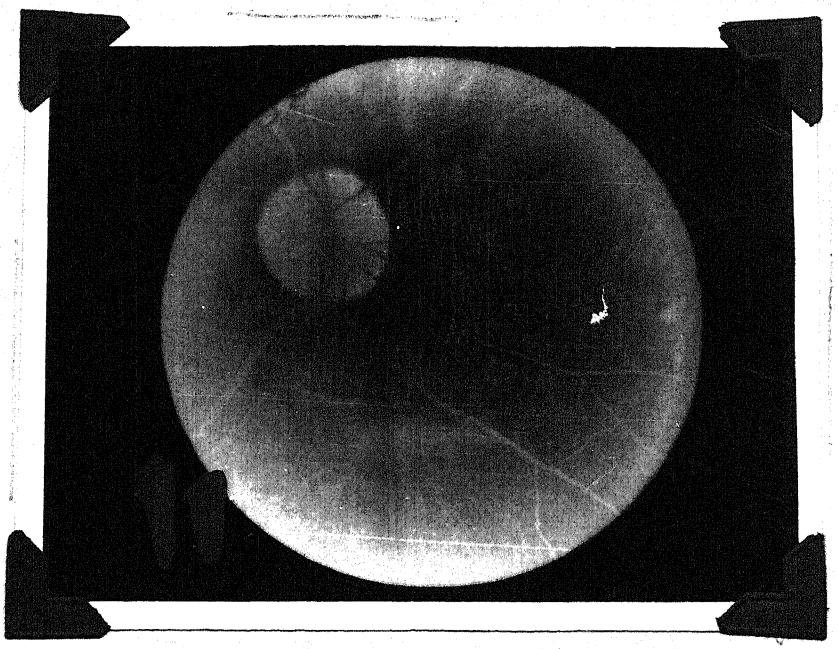
- COMPLETE FLUORESCENT DISC.
- STAINING CRESCENT AREA.
- DIFFUSE MOTTLING OF CHOROIDAL FLUORESCENCE.
- RARIFIED CAPILLARY NET WORK.



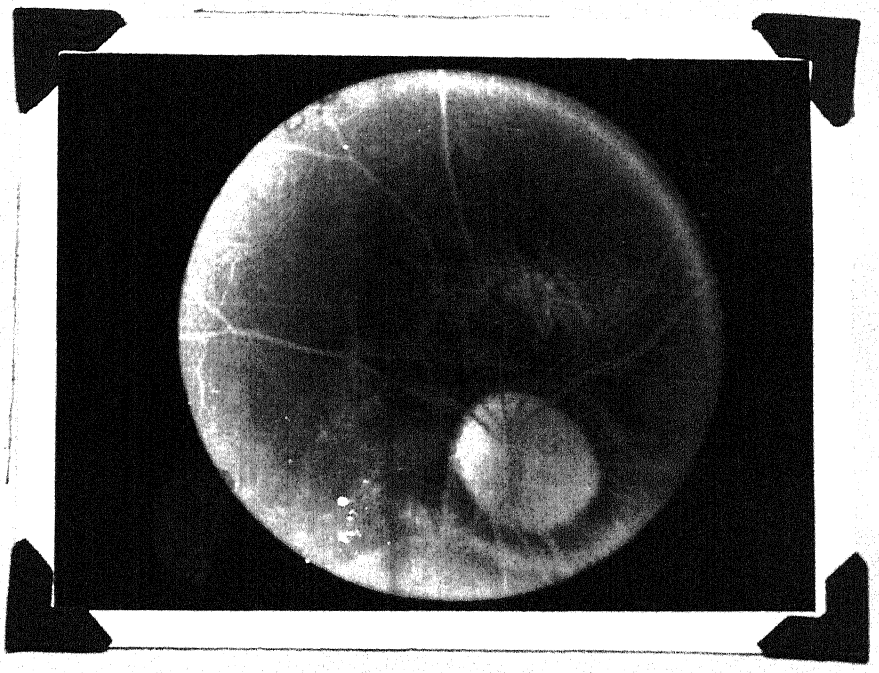
ARTERIAL PHASE OF FLUORANGIOGRAM SHOWING, NO DYE IN SOME VEINS WHILE IN SOME LAMINAR PATTERN OF FLOW, VARIOUS ISCHEMIC ZONES & CHORIORETINAL ANASTOMOSIS IN CRESCENT AREA.



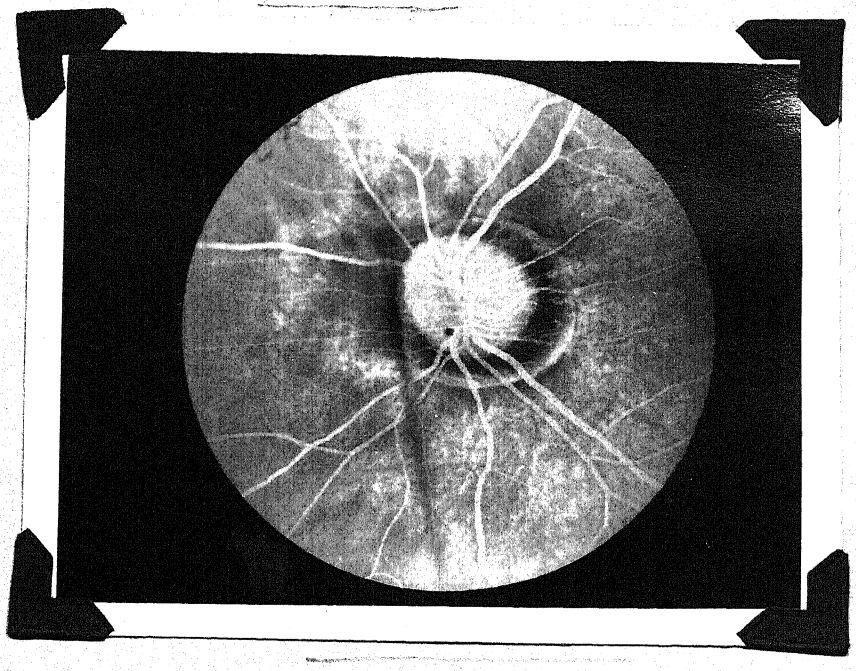
FLUORANGIOGRAM OF ARTERIO-VENOUS PHASE SHOWING-CHOROIDAL VESSELES AND CHORIORETINAL ANASTOMOSIS, CIRCLE OF ZINN, JUXTAPAPILLARY ATROPHY & INLARGED, DARK MACULAR AREA.



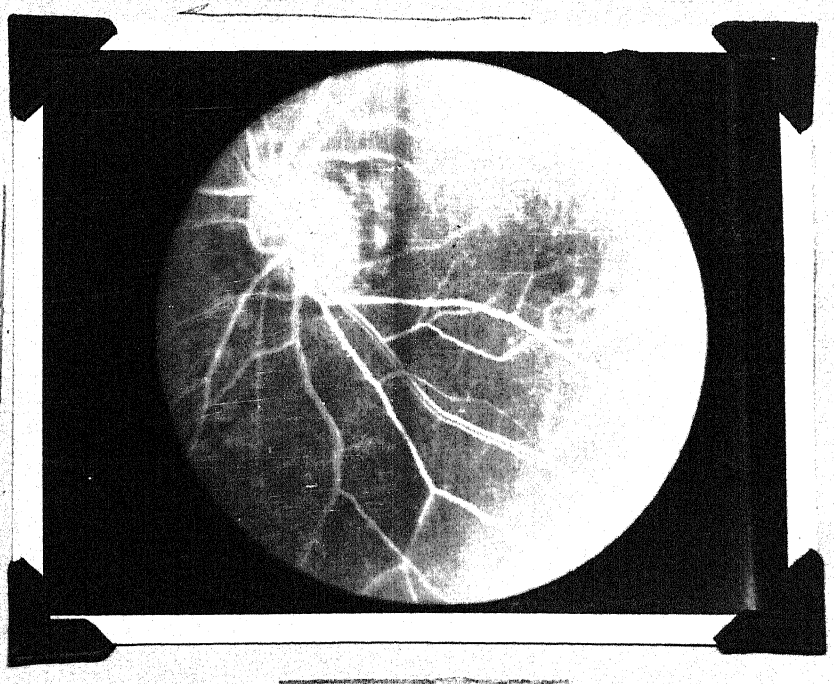
ANGIOGRAM REVEALING EMPTY CHOROIDAL VESSELES EVEN IN THE EARLY ARTERIOVENOUS PHASE, COMPLETE DISC FLUORESCENCE & PERIPAPILLARY ATROPHY.



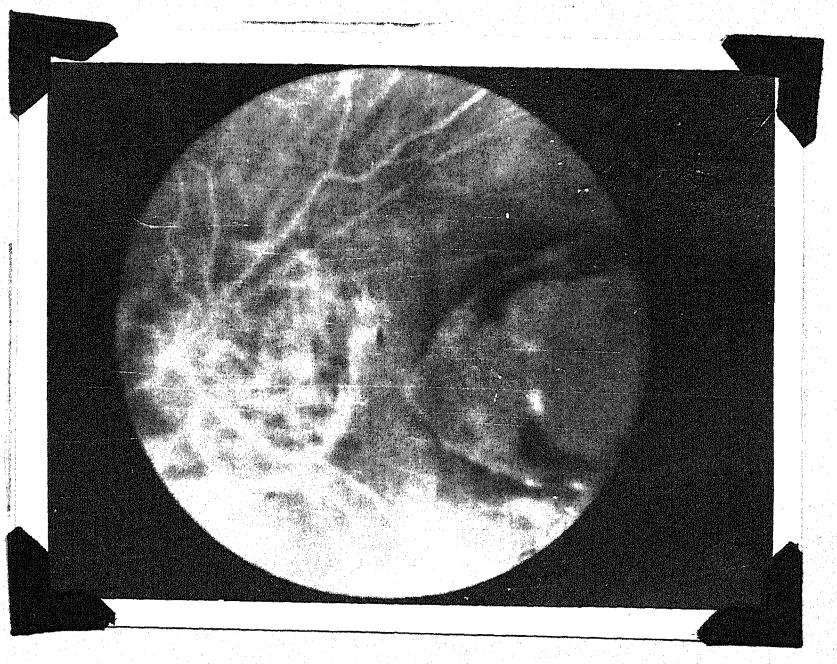
FLUORESCEIN ANGIOGRAM SHOWING COMPLETE DISC FLUORESCENCE, HYPERFLUORESCENT PATCH SURROUNDED BY VARIABLE DENSITY OF HYPOFLUORESCENCE SUPERIOTEMPORALLY, PERIPAPILLARY CHOROIDAL ATROPHY.



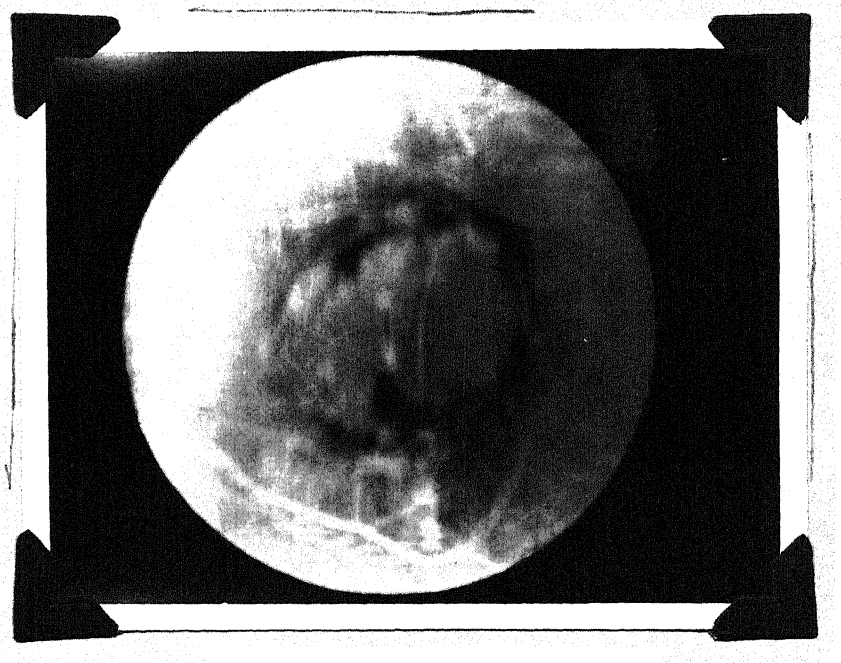
ANGIOGRAM SHOWING, COMPLETE DISC FLUORESCENCE, EPIPAPILLARY CAPILLARIES CHORIORETINAL ANASTOMOSIS & PERIPAPILLARY CHOROIDAL ATROPHY.



ANGIOGRAM SHOWING COMPLETE DISC FLUORESCENCE, ANNULAR CRESCENT, LOCALIZED HYPERFLUORESCENCE IN PARAMACULAR AREA IN EARLY ARTERIOVENOUS PHASE.



FLUORESCEIN ANGIOGRAM REVEALING CIRCUMPAPILLARY ATROPHY, WIDE ATROPHIC PATCH IN MACULAR & PARAMACULAR AREA.



FLUORANGIOGRAM SHOWING TWO HYPERFLUORESCENT PATCH MATTING TOGETHER WITH PERIPHERAL BLOCKED FLUORESCENCE IN LATE ARTERIOVENOUS PHASE OF THE DYE TRANSIT.

DISCUSSION

DISCUSSION

It is a well known observation that eyes having high myopia have a poor visual acuity on account of wide spread degenerative changes in the central fundus. A number of workers have described the ophthalmoscopic changes in the central fundus, specially those having pathological myopia (Salzmann, 1902; Lloyd, 1953; Curtin and Karlin, 1971). Areas of chorioretinal atrophy are most commonly seen in the region of the posterior pole (Curtin and Karlin, 1971). However, no satisfactory explanation is available to account for the development of such degenerative changes in the myopic eyes. Lately, a few investigators have tried to correlate the occurrence of degenerative changes in highly myopic eyes with fluorescein fundus angiography (Khasanova and Taldaeva, 1975; Stangos et al, 1976; Yoshihara et al, 1976; Watanabe et al, 1976; Levy et al, 1977; Yoshihara, 1976; Hayashi et al, 1980; Giovnnini et al, 1980; Hoyt C.S. et al, 1981; Michael Shapiro et al, 1985). However, fluorescein angiographic observations are only in isolated reports and throw some light on the changes in the myopic eyes. The reports are far from satisfactory. The present study is an attempt to study the changes in myopia both ophthalmoscopically and fluorangiographically.

CHOROIDAL CIRCULATION

In the normal eyes (Group I), a typical uniform choroidal fluorescence was observed on the temporal and the nasal quadrants. This is due to a healthy choroidal vascular bed and a structurally intact normal pigment epithelium.

It is the pigment epithelium which in the presence of healthy choriocapillaris bed obscured individual choroidal vessels and is responsible for a homogeneous diffused background fluorescence in the normal eyes. This is further proved by the fact that in eyes having an absence of, or lighter pigments, as in albinotics or high myopia(Archer et al, 1970) or following pathological disease process (Hayreh, 1974), there is less interference with the choroidal fluorescence which could be seen even in the earlier phase of dye transit (Schatz et al, 1978). Thus it would be quite reasonable to believe that the pigment epithelium is a barrier between the choroid and the retina which normally hinders with the ophthalmoscopic visualisation of choroidal vascular bed.

In group II, the majority of the eyes, having a low myopic refractive error, showed a typical uniform choroidal fluorescence even in the early arterial phases, while in the remaining eyes it was seen to begin initially from the temporal specially superotemporal quadrants near the disc and eventually became uniform in later phases of the dye transit. This observation clearly suggests that in

eyes having low degree of myopia, the choroidal vascular bed is more or less uniformly healthy and patent. A functionally active choroidal vascular bed on the temporal side in these eyes may be one of the reason for a relative absence or macular, paramacular and centrocaecal degenerative lesions in such eyes. In other words, a healthy choroidal circulation in eyes with low myopia may impart an immunity to these eyes towards development of degenerative lesions.

On the other hand, a very interesting phenomenon was observed in eyes having pathological myopia (Group III). In 11(73.3%) out of 15 eyes, a characteristic nasal choroidal fluorescence was observed. In 4(26.7%) eyes, out of this 11 eyes no temporal fluorescence was observed at all while in rest of the eyes, nasal choroidal fluorescence was more marked. Only in 4(26.7%) eyes choroidal fluorescence was uniform.

This finding of predominant nasal choroidal fluorescence and complete absence of temporal fluorescence even in the late phases of the dye transit, clearly suggests that there is a structural defect in the temporal choroidal vasculature which may be affected from mild to severe degree. Since, the outer layers of the retina derive their blood supply from the choroidal bed, it would be quite understandable that an ischaemic choroidal bed in eyes having pathological myopia is the causative factor for the

development of various lesions in the central and peripheral fundus. In man, the watershed zone between the medial posterior ciliary artery and lateral posterior ciliary artery usually lies between the optic disc and the macular region. It has also been observed that watershed zone is highly vulnerable to ischaemic disorders (Hayreh, 1976). This further proves that the temporal part of the choroid, like macular region is more vulnerable to the ischaemia. Further, the elongation of the eye ball in the anteroposterior diameter puts a burden on the choroidal bed on the posterior pole and eventually resulting in a marked chorioretinal atrophy (Curtin and Karlin, 1971). It has also been observed in the study that the calibre of the large sized choroidal vessels was found to be lesser in eyes having high pathological myopia (-18.0 D spherical to -24.0 D spherical) than eyes having comparatively lesser degree of pathological myopia (-9.5 D spherical to -16.0 D spherical). These findings, therefore, suggest that the choroidal vascular bed plays a very important role in the integrity of the retina and once this is compromised, the degenerative changes appear. Similar observations have also been made by Balshnova (1978).

PIGMENT EPITHELIUM DEFECTS AND CHOROIDAL VESSEL SCLEROSIS

In 11(73.3%) out of 15 eyes (Group III), individual choroidal vessels were observed in all the phases of the dye transit. A uniform filling of choroidal vessels was

seen in 6(20.0%) out of these eyes and no empty vessels was observed. In the remaining eyes, this feature was not uniform so much so that a few of the individual choroidal vessels could be seen free from fluorescein in early and late fluorangiograms. This would suggest that there is a marked obliteration of choroidal vessels and choriocapillaris in eyes having high pathological myopia and this also proves the role of underlying choroidal deficit in the causation of degenerative changes. The visualisation of individual choroidal vessels is due to a defective overlying retinal pigmentary epithelium having insufficient pigment to block the background fluorescence. Relative destruction of choriocapillaris alongwith the defective overlying pigment epithelium may be another factor for the visualisation of the larger choroidal vessels. Archer et al (1970) observed that the large or intermediate sized choroidal vessels become visible on fluorangiograms when the choriocapillaris are absent. In areas where the retinal pigmentary epithelium is completely absent the fluorescence is marked upto the late phases of the dye transit. Hyperfluorescence in these areas represents the leakage of the dye from the choriocapillaris in the early retinal venous phase and as scleral staining in the late phases. In contrary to this, the collection of pigmentary clumps in one eye caused the hypofluorescence in the same region

showing an obscured choroidal fluorescence throughout all the stages of the dye transit. The similar findings have been reported by Rosen (1969).

DISC FLUORESCENCE

Although, the temporal pallor of the disc in some eyes was the commonest finding on ophthalmoscopic examination, yet the fluorescein angiography revealed a complete filling of the disc in all the three groups. In these eyes the initial filling of the disc was seen to commence from the temporal side, which gradually increased and finally the whole disc revealed a generalized and uniform fluorescence. The initial filling of the disc is on account of increased vascularity of the disc on the temporal than on the nasal side. This has also been comfirmed and reported by Hayreh (1974). It is quite important to know this fact as on ophthalmoscopic examination the temporal half of the disc appears comparatively paller than of the nasal half(Hayreh, 1974). This is probably due to the presence of glial tissue on the surface of the disc, which is more on the temporal side and covers the capillaries of the disc and is responsible for a relative pallor of the disc. Yoshihara et al, (1976) believe that such a reactive glial proliferation is an important factor for the appearance of the pallor of the disc in high myopic patients. It would thus appear that the colour of the disc as seen by ophthalmoscopic observations in myopic individuals does

not correlate with the vascularity in this region. It is therefore, seen that the disc is relatively normal in myopic eyes.

DISC CRESCENT

An important finding in myopic eyes is the presence of a crescent mainly on the temporal side. In the present study the disc crescent was typically, seen on the temporal side in a large majority of the eyes having simple and pathological myopia. The fluorescein angiography in earlier arterial phase reveals a fluorescence at the extreme temporal margin of the crescent which gradually increased with the passage of the dye so that in the late venous phase, the entire area of the crescent revealed a uniform fluorescence on account of staining of the sclera. The uniform fluorescence in late phases is because of scleral staining, is proved by the fact that in the area of the crescent, the choroid and pigment epithelium are absent (Duke Elder, 1970). This late fluorescence in the crescent area often creates difficulty in identifying the crescent from the disc margin and seen to merge with one another. However, in the early arterial phase, the crescent area is well defined.

Another interesting feature in the myopic eyes is the presence of vascular branches from the peripapillary choroidal plexus or circle of zinn in the crescent area.

These vascular branches are normally not visible on ophthalmoscopic examination and fluorescein angiography often reveals an anastomosis between these vessels and the branches of the retinal vessels over the crescent area. Such a feature was seen in 4(23.5%) eyes in simple myopia and 5(33.3%) eyes in pathological myopia. The presence of vessels in an isolated or a plexus like pattern in the crescent area has also been reported by Stangos et al (1976). The presence of a chorioretinal anastomosis in eyes with pathological myopia may be quite significant as it may play an important role in maintaining the nutrition of the retina to an optimum level, even in the presence of choroidal sclerosis. Thus it is presumed that the presence of such an anastomosis would play a compensatory role in maintaining the circulation to a reasonably normal status. In eyes where such an anastomosis was present, the degenerative changes in the central area were minimal and the visual acuity was comparatively better than the eyes where such a feature was not present even when the refractive error in both the groups was nearly similar.

It is not unusual to find a cilioretinal artery arising from the temporal edge of the disc and running upto the macular area. Fluorescein angiography reveals that this vessel originates from behind the disc and is seen on the disc margin with a bend. Further the filling of this vessel occurs simultaneously with the choroidal

filling and since it fills earlier than the retinal vessels, it is believed to arise from the peripapillary choroidal plexus (Archer et al, 1970). Stangos et al (1976) have also observed this vessel to arise from the peripapillary choroidal plexus in the crescent area.

PERIPAPILLARY REGION

The peripapillary region in high myopia has been studied by a few workers with the help of fluorescein angiography (Stangos et al, 1976) Yoshihara et al, 1978; Ichata et al, 1978). Stangos et al (1976) have reported a dense net work of peripapillary choroidal vessels formed by radially arranged branches in high myopia. Amalric (1969) has reported the presence of peripapillary choroidal plexus in concentric circles. In the present study the peripapillary region except in the crescent area in simple myopia (Group II) did not reveal any demostrable pathology. This would suggest that in individuals having low myopic refractive error, the retinal pigmentary epithelium in the peripapillary region as elsewhere is healthy and intact and therefore explains the absence of a visible peripapillary choroidal fluorescence, plexus or atrophy in this region. On the contrary, in individuals having high pathological myopia, the peripapillary retinal pigmentary epithelium is relatively unhealthy with less pigment (Rosen, 1969), and is thus responsible for the occurrence of visible peripapillary choroidal vessels or plexus. In the present study, peripapillary choroidal plexus was seen in 3(20.6%)

eyes.

A very important observation in pathological myopia is the presence of peripapillary choroidal atrophy. In the present study, it was seen either in a circumpapillary or localised form. The fluorescein angiography in some eyes revealed a diffused hyperfluorescence in the area of circumpapillary atrophy in the late phases, while in some more eyes, a minimal marginal fluorescence even in the late phases was observed in these atrophic areas. It is probable that the absence of fluorescence in the area of peripapillary atrophy even in late phases, is on account of a relative atrophy and sclerosis of choroidal vascular bed. Schatz et al, (1978) have observed that the hypofluorescent area in late phase in the region of circumpapillary atrophy is due to the vascular filling defect of the choriocapillaris and hyperfluorescent rim in later phases of the angiograms is due to leakage into the area from the adjacent normal choriocapillaris. It is further possible that the number of sclerosed choroidal vessels having no fluorescein in their lumen may be more in this area in such eyes and may hinder with the visualisation of the scleral staining in the late phases. On the other hand, eyes showing a diffused fluorescence in the area of circumpapillary atrophy have a relatively healthy choroidal vasculature in this region. Further it is interesting to note that eyes showing diffuse fluorescence in this area have a lesser degree of myopia while eyes not showing this feature, had a high refractive

error. This would again prove the presence of a relative sclerosis and obliteration of choroidal vessels in the area of peripapillary choroidal atrophy as elsewhere in the eyes having pathological myopia.

RETINAL CIRCULATION

It has been observed by few workers that myopia has an adverse effect on the calibre of the retinal vessels and this can be one of the additional factors in the development of degenerative changes in high myopia (Khasanova and Taldaeva, 1975). In the present study, the calibre of both the branches of the retinal artery and vein near the disc border was found to be narrower in myopic eyes, more so in pathological eyes than that of normal eyes.

The calibre of the retinal artery branches in eyes belonging to normal (Group I) , simple myopia (Group II) and pathological myopia (Group III) were found to be in the ratio of 1:0.73:0.67 respectively, while the ratio of vein tributaries at the same site was found to be in the ratio of 1 : 0.69 : 0.68 respectively. This would further suggest that the narrowing of the retinal vessels in high myopia is another factor responsible for relative retinal ischaemia and predisposing the degenerative changes. A decreased calibre of retinal vessels in high myopia has also been reported by Khasanova and Taldaeva (1975).

MACULAR REGION

The macular region in myopia has aroused the

maximum interest in many ophthalmologists because of a number of degenerative changes seen which in turn are responsible for poor visual acuity in majority of the eyes.

In normal eyes and eyes with low myopia the fluorescein angiography does not reveal background choroidal fluorescence in the macular area on account of an intact retinal pigment epithelium. The dark appearance of macula on fluorescein fundus angiography in such eyes is due to retinal capillary free zone around fovea and rich concentration of Zanthophyll and lipofuschin pigments in the pigment epithelial cells in this area(Schatz et al, 1978). Cilioretinal artery also contributed to the nourishment of the parafoveal region in three eyes.

On the other hand in high pathological myopia where the retinal epithelium is comparatively unhealthy, fluorescein angiography reveals a number of findings. The commonest is an area of localised or diffused macular or paramacular hyperfluorescence as seen in this study. The patchy absence of retinal pigment epithelium would also explain the visualisation of choroidal sclerosis and choroidal atrophy in the macular and paramacular area. Interesting finding on fluorescein angiography in the present study was the presence of an irregular area of hyperfluorescence, surrounded by a nonfluorescent zone in 2(13.3%) eyes out of 3 such eyes. In 1 eye, however, the nonfluorescent zone was not seen. Ophthalmoscopically, this

area was seen as an irregular dark patch in the macular area (Fuch's spot) obscuring the underlying details. A number of workers have reported the occurrence of subretinal neovascular membrane (SRNM) in the macular area underlying Fuch's spot (Levy et al, 1977; Schatz et al, 1978). However, in the present study, such a feature was not observed, although the patchy hyperfluorescence as seen in the eyes could be due to leakage of the dye from the underlying choriocapillaris. In one eye the associated retinal pigmentary epithelium defect on either side of the Fuch's spot contributed to the late hyperfluorescence in this region. Yoshihara (1979) strongly feels that subretinal neovascular membrane formation is one of the characteristic feature of Fuch's spot. Undoubtedly the presence of Fuch's spot in the macular area is one of the important cause for the marked visual impairment. Lacquer Crack Lesions as observed ophthalmoscopically (Klein et al, 1975) and fluorangiographically (Watanabe et al, 1976; Yoshihara, 1979) were, however, not seen in the present study. This lesion is presumed to be composed of Bruch's membrane, and appears on fundus examination in the form of irregular yellow lines in the macular and paramacular area.

To conclude, it is emphasized that anatomically and functionally active choroidal vasculature plays a very important role in maintaining the integrity of the retina in normal eyes. In conditions including high myopia where the underlying choroidal vascular architecture is

relatively weak, the retinal nutrition suffers and consequently degenerative changes develop. The consequences of the obliteration of choroidal vasculature are very grave in the macular area which suffers irreversible damage and is frequently responsible for a visual impairment, as seen in high myopia.

SUMMARY AND CONCLUSION

SUMMARY & CONCLUSION

Since the introduction of this technique by Novotny and Alvis (1961), a number of workers have described the clinical application of this procedure in various ocular disorders(Hayreh, 1969, 1972, 1974; Hayreh and Baines, 1972). Very few workers have described the utility of this procedure in myopic individuals (Stanges et al, 1976; Yoshihara et al, 1976; Levy et al, 1977; Yoshihara, 1979; Avetisove et al, 1977; Giovannini et al, 1980; Hoyt et al, 1982; Michael Shapiro et al, 1985).

The present study was therefore undertaken to study the anatomical changes in myopic eyes fluorangiographically and to correlate these changes with the ophthalmoscopically demonstrable degenerative changes in the fundus. The study was undertaken on 38 eyes of 26 subjects by fluorescein fundus angiography.

The eyes so studied were divided into three groups.

Group - I (Normal eyes-6)

This consisted of 6 emmetropic eyes of 4 individuals without any demostrable pathology either in the anterior segment or in fundus.

Fluorescein angiography revealed a uniform choroidal fluorescence in all the eyes in the early arteriovenous phases. The disc showed an initial temporal fluorescence which later on became uniform so that in late venous phase the whole disc revealed a generalized fluorescence. Cilioretinal artery was seen in 2(33.33%) eyes.

The macular area in these eyes appear darker obscuring the underlying choroidal fluorescence and no pathology was noticed.

The average calibre of the branches of central retinal artery and vein near the disc border was observed & compared with group II & III.

Group - II (Simple myopia - 17 eyes)

These eyes were considered to have simple myopia because of the presence of minimal degenerative changes in the fundus. The macular area was normal. Anterior segment was also normal and the best corrected visual acuity was 6/6 (11 eyes 64.70%) and up to 6/24 (6 eyes - 35.30%). Family history of myopia was noted in 7(58.3%) individuals.

Fluorescein angiography revealed a characteristic uniform choroidal fluorescence in these eyes without any visible choroidal vessels. The disc filling was seen to commence from the temporal side, which gradually became

uniform. The temporal crescent was the commonest finding and consistently revealed a late staining which was clearly seen in late phases. The choroidal vessels from the peripapillary choroidal plexus or circle of Zinn underlying the crescent area were observed in 4(23.5%) eyes which also revealed a chorioretinal anastomosis in the crescent area. The peripapillary region did not reveal any pathology.

The macular area appeared darker, obscuring the underlying choroidal fluorescence and no pathology was detected in this region. Cilioretinal artery, supplying upto the parafoveal region was observed in one eye.

The average calibre of the branches of the central retinal artery and vein near the disc border was found to be less than group I and more than that of group III.

Group - III (Pathological myopia - 15 eyes)

There was a history of rapid increase of myopia in all the individuals. The prominent ophthalmoscopic findings in these eyes were in the form of prominent disc crescent, peripapillary choroidal atrophy, chorioretinal atrophy and extensive choroidal sclerosis, macular degeneration and Forster Fuch's flecks. The anterior segment in these eyes was normal except for the presence of lenticular and vitreous opacities in 1 eye. The

corrected visual acuity varied from 3/60 to 6/24. A family history of myopia was noted in 8(80%) individuals.

Fluorescein fundus angiography revealed an initial nasal choroidal fluorescence in 11(73.3%) eyes, and became uniform in all the eyes except in 4(26.7%) eyes where no temporal choroidal fluorescence was seen even upto the late venous phase. In 12(80.0%) eyes, the individuals choroidal vessels were observed. In 5(33.3%) out of these 11 eyes, few of the individual choroidal vessels could be identified free from fluorescein even in early arteriovenous angiograms. 'The average calibre of large sized choroidal vessels was 1 mm (8 times magnification of the negative) in eyes having low refractive error ranging from -9.5 D spherical to -16.0 D spherical and 0.5 mm (8 times magnification of the negative) in eyes having high refractive error ranging from -18.0 D spherical to -24.0 D spherical'.

The disc fluorescence was complete. A well defined disc crescent predominantly located on the temporal side was noted in 10(66.7%) eyes. The peripapillary region showed circumpapillary atrophy (5 eyes) and juxtapapillary atrophy (1 eye). In 2(13.3%) eyes a late staining in the area of circumpapillary atrophy was observed while in rest 3(20.0%) eyes, no staining was observed in the late phases of dye transit. The choroidal vessels from the peripapillary choroidal plexus or circle of Zinn underlying the crescent

area and circumpapillary choroidal atrophy were observed in 5(33.3%) and 5(33.3%) eyes respectively.

Paramacular area revealed a localized or patchy hyperfluorescence in 1 eye, while a diffused hyperfluorescence was found in 1 eye. In 3(20.0%) eyes showing an irregular darkish black patch (Forster Fuch's flecks) ophthalmoscopically, fluorescein fundus angiography revealed an irregular area of hyperfluorescence surrounded by a nonfluorescent zone in 2 eyes. Sclerosed choroidal vessels were seen in one eye. The extensive choroidal atrophy with pigmentry defect was also seen in one eye. In 4 eyes, although ophthalmoscopic examination revealed a dull foveal reflex and pigment mottling, fluorescein angiography did not reveal any pathology.

The calibre of the branches of central retinal artery and vein near the disc border was found to be narrowest in this group.

Late staining in the area of wide pigmentary epithelium defect in macular area (1 eye) and a hypofluorescent patch in the area of pigmentary clump (1 eye) were also observed.

In general, it was therefore, observed that eyes with simple myopia revealed no changes fluorangiographically while eyes with pathological myopia show defective choroidal vascular bed on the temporal side of the disc, choroidal sclerosis, pigmentary epithelium defects, peripapillary

choroidal atrophy and changes in the macular and paramacular region. The gravity of the changes were seen more clearly on fluorescein fundus angiography than on ophthalmoscopic examination.

CONCLUSIONS

On the basis of the observations made in the present investigation, the following conclusions have been drawn:

1. Myopic eyes specially those with high myopia are prone for certain structural defects which are responsible for the occurrence of degenerative changes in the fundus.
2. Retinal pigment epithelium is a barrier between the choroid and retina and when intact and healthy, hinders with the ophthalmoscopic and fluorangiographic visualization of choroidal vascular bed in normal and simple myopic eyes.
3. In eyes with pathological myopia retinal pigment epithelium is anatomically defective and thus is responsible for ophthalmoscopic as well as fluorescein angiographic visualization of various degenerative lesions like choroidal sclerosis and choroidal atrophy.
4. The choroidal vascular architecture in eyes with pathological myopia is defective in general and in particular it is grossly obliterated and sclerosed in the temporal part.

5. Choroidal vascular obliteration is the single most important factor in the causation of various degenerative changes in eyes with pathological myopia as a result of ischaemia.

6. The obliteration and sclerosis of choroidal vessels is directly proportional to the increase of myopic refractive error.

7. Temporal pallor of the disc in myopic eyes as seen ophthalmoscopically is a deceptive phenomenon and does not reflect a low vascularity in this region. On the contrary temporal part of the disc in such eyes is more densly supplied by capillaries as revealed by fluorescein fundus angiography.

8. Temporal crescent is the commonest finding in myopic eyes, the crescent area shows a uniform fluorescence in late phase of dyetransit on account of staining of sclera, often making difficult to differentiate it from the disc margin.

9. Vascular branches from the peripapillary choroidal plexus or circle of Zinn are prominently seen after fluorescein angiography either at the margin or in the crescent area or in the area of peripapillary choroidal atrophy in myopic eyes specially in eyes with pathological myopia.

10. Anastomosis between choroidal branches from the peripapillary choroidal plexus or circle of Zinn and

branches of retinal artery is seen in the crescent area in eyes with simple myopia and also in the area of peripapillary choroidal atrophy in pathological myopia.

11. Presence of a chorioretinal anastomosis in myopic eyes beneficial in maintaining the integrity of retinal vasculature even in the presence of underlying choroidal sclerosis.

12. The peripapillary region in eyes with pathological myopia commonly shows choroidal atrophy, most commonly in the form of circumpapillary and farely as juxtapapillary choroidal atrophy.

13. The underlying choroidal vasculature in the area of peripapillary choroidal atrophy is usually sclerosed in eyes having a high pathological myopia and thus would account for absence of late fluorescence in this region. However, in eyes with a lesser degree of pathological myopia the choroidal sclerosis may be relatively less and therefore a uniform late choroidal fluorescence is seen in such eyes.

14. The retinal vessels are comparatively narrwer in myopic eyes than normal ones and this together with choroidal obliteration would account for most of the degenerative changes in such eyes.

15. The macular area in eyes with simple myopia does not show any abnormality either ophthalmoscopically or on fluorescein angiography. On the other hand in

pathological myopia where retinal pigment epithelium is defective it reveals various findings prominent of these being choroidal sclerosis and choroidal atrophy.

16. The feature of Forster Fuch's flecks which appears ophthalmoscopically as a darkish black area in the macular area reveals characteristic central fluorescence suggestive of dense choroidal vasculature surrounded by a nonfluorescent zone which suggests presence of defects in retinal pigmentary epithelium.

BIBLIOGRAPHY

BIBLIOGRAPHY

Allen, L., Kirkendall, W.B., Snyder, W.B. and Frazier, O. (1966): Arch. Ophthal. 75 : 192.

Amalric, P. (1971): Circulation choridienne. In : P. Amalric (ed.), Fluorescein angiography. Proceedings of the International Symposium on Fluorescein Angiography. 192-203, Karger(1971).

Anderson, D.R. and Dvais, B.B.(1976): Arch. Ophthal. 92 : 422.

Archer, D.B. (1972): Fluorescein Angiography, In : Allen, E. Krill(ed.), Hereditary Retinal and Choroidal Diseases, Vol. I, Evaluation. Harper and Row Publishers, New York, 1972, pp. 83, 68, 89, 96, 160, 96, 97, 138, 139.

Archer, D., Krill, A.E. and Newell, F.W. (1970): Amer. J. Ophthal. 69 : 543.

Archer, D.B., Krill, A.E. and Newell, F.W. (1971): Amer. J. Ophthalmol. 71 : 266.

Avetisov E.S., and Savitskaya N.F.: Ann. Ophthal. 2 : 1261.

Balshanova, A.B. (1978): Vestn. Oftal. 95 : 25.

Charamis, J.N., Katsourakis, G. Mandras (1966): Amer. J. Ophthalmol. 61 : 1078.

Corrado-Balacco Gabrieli. : Ann. Ophthal., 15 : 313.

Crock, G.W.(1970) : Trans. Ophthal. Soc. U.K. 90 : 577.

Curtin, B.J. and Karlin, D.B.(1971) : Amer. J. Ophthalmol. 71 : 42.

Delori, F. and Ben-Sira-I (1975) : Invest. Ophthalmol. 14 : 487.

Dollery, C.T., Hodge, J.V. and Engel, M.(1962) : Brit. Med. J. 2 : 1210.

Donaldson, D.D. (1964) : Amer. Ophthal. Soc. 62 : 430.

Duke Elder, S. and Abrams, D. (1970) : Ophthalmic optics and Refraction. In: Duke Elder, S. (ed.). System of Ophthalmology. Vol. V. St. Louis, C.V. Mosby, 1970 : 324.

Ernest, J.T. and Archer, D. (1973) : Amer. J. Ophthalmol. 75 : 973.

Evans, P.Y., Shimizu, K., Limaye, S., Deglin, E. and Wruck, J. (1974) : Arteriolar flow studies by retinal Cineangiography. Fluorescein Angiography. p. 27. Tokyo, Igaku Shoin Ltd. 1974.

Fehman, S.S., Schaffer, C. and Van Heuven, W.A.J. (1976) : Advances in TV-Fluorangiography. In: Lacy, J.J.D. (ed.) : Internation Symposium on Fluorescein Angiography. Doc. Ophthal. Proceeding Series. Dr.W.Junk B.V. Publishers, The Hague, 1976, Vol. 9 : 19.

Ferrer, O. (1965) : Amer. J. Ophthalmol. 60 : 587.

Farrer, O. (1974): Arch. Soc. Amer. Oftal. Optom., 10 : 119.

Flower, R.W.(1972): Amer. J. Ophthalmol. 74 : 600.

Flower, R.W.(1973): Invest. Ophthal. 12 : 881.

Frazier, O. and Allen, L. (1972): In: "Current Concepts in Ophthalmology", Vol. 3. F.C. Blodi, Mosby, St. Louis, pp. 41-47.

Gas, J.D.M. (1967): Am. J. Ophthalmol. 31 : 689.

Giovannini, A., Colombati, S., Ciliberti, C. (1980): Doc. Ophthal. Proceeding Series, 28, 'IIIrd International Conference on myopia'.

Gass, J.D.M.(1973): Trans. Am. Acad. Ophthal. & Otolarying. 77 : 310.

Gitter, K.A., Housen, B.P., Sarin, L.K. and Justice, J. (1968): Arch. Ophthal. 80 : 449.

Haining, W.M. (1966): Br. J. Ophthal. 50, : 587.

Haining, W.M. and Lancaster, R.C. (1968): Arch. Ophthal. Chicago). 79 : 10.

Hart, L.M., Heyman, A., Linhart, J.W., McIntosh, H. and David, N.J. (1963): J. Lab. Clin. Med. 62 : 703.

Hayashi, K., Uchida, A., Fukushita, K., Takizawa, E. and Tokora, T. (1980): Folia Ophthal. Japan, 31 : 459.

Hayreh, S.S. (1968): Brit. J. Ophthal. 52 : 566.

Hayreh, S.S. (1969): Brit. J. Ophthal. 53 : 721.

Hayreh, S.S. (1972): Brit. J. Ophthal. 56 : 175.

Hayreh, S.S. (1973): Trans. Amer. Acad. Ophthal. & Otolaryng. 77 : 300.

Hayreh, S.S. (1974): Brit. J. Ophthal. 58 : 391.

Hayreh, S.S. (1975): Brit. J. Ophthal. 59 : 631.

Hayreh, S.S. (1976): Physiological Anatomy of the Choroidal Circulation. In: Laey, J.J.D.(ed.). International Symposium on Fluorescein Angiography. Doc. Ophthal. Proceeding Series. Dr. W. Junk Bv Publishers, The Hague (1976), Vol. 9 : 201.

Hayreh, S.S. and Baines, J.A.B. (1972): Brit. J. Ophthal. 56 : 719.

Hayreh, S.S. and Baines, J.A.B.(1973): Brit. J. Ophthal. 57 : 217.

Hayashi, K., Uchida, A., Fukushita, K., Takizawa, E., Tokoro, T. (1980) : Folia Ophthalmol. Jph. 31 : 459.

Hendrickson, P., Oniki, S. and Elliott, J. (1970) : Arch. Ophthal. 83 : 580.

Hickam, J.B. and Frayser, R. (1965): Invest. Ophthal. 4 : 876.

Hill, D.W. and Young, S. (1976): Cine Angiographic inflow measurements using fluorescein and Indocyanine green. In: Laey, J.J.D. (ed.), International Symposium on Fluorescein Angiography. Doc. Ophthal. Proceeding Series. Dr. W. Junk B.V. Publishers, The Hague, 1976. Vol. 9 : 67.

Hisatomi, Ch. and Suzuki, Y.(1974) : Simultaneous bilateral angiography. Proc. Int. Symp. Fluorescein Angiography. Tokyo, 1972, Igaku Shoin Ltd., Tokyo : 269.

Hodge, J.V. and Clemett, R.S. (1966): Amer. J. Ophthal. 61 : 1400.

Hoyt, C.S., Stone, R.D., Fromer, C., et al. (1981): Amer. J. Ophthal. 19 : 197.

Hyvarinen, L., Naumenne, A.E., George, T. and Weinstein, O. (1969): Amer. J. Ophthalmol. 67 : 653.

Hyvarinen, L., Naumenne, A.E., Kelley, J. and Cantellino, S. (1971): Amer. J. Ophthalmol. 71 : 17.

Hyvarinen, L. and Nieminen, H. (1967): Acta Ophthal. (Kbh.). 45 : 100.

Jensen, Viggo A. (1973): Acta Ophthalmological. 51 : 583.

Jensen, V.A. and Lundbaek, K. (1968): Acta Ophthal. (Kbh.), 46 : 584.

Justice, J. Jr. and Lehman, R.P. (1976): Consideration of the cilioretinal circulation. In: Laey, J.J.D.(ed.). International Symposium on Fluorescein Angiography. Doc. Ophthal. Proceeding Series. Dr. W. Junk B.V. Publishers, The Hague, 1976, Vol. 9 : 261.

Khasanova, N.Kh. and Taldaeva, A.Kh. (1975): Vestn. Oftal. 22 (1) : 49.

Klein, R.M. and Curtin, B.J. (1975): Amer. J. Ophthalmol. 79 : 386.

Kogrus, K. and Chromokos, E. (1969): J. App. Physiol. 26 : 154.

Kogrus, K., David, N.J., Yamamouchi, U. and Chromokos, E. (1970): Arch. Ophthal. 83 : 209.

Kooijman, A.C.(1972): Ophthalmologica. 164 : 398.

Kuwamoto, K. (1971): Fluorescence fundus photography by acridine orange. Proc. Int. Symp. Fluorescein Angiography, Albi 1969, 12, Karger, Basal (1971).

La Piana, F.G. and Penner, R. (1965): Arch. Ophthal. 79 : 161.

Laux, U.R.(1976): Circulation parameters comparison on both eyes by simultaneous fluorescein angiography. In: Lacy, J.J.D. (ed.), International Symposium on Fluorescein Angiography. Doc. Ophthal. Proceeding Series. Dr. W. Junk, B.V. Publishers, The Hague, 1976, Vol. 9 : 39.

Levy, J.H., Pollock, K.M. and Curtin, B.J. (1977): Ann. Ophthalmol. (Chic.), 9 : 1433.

Lloyd, R.L.(1953): Trans. Am. Ophthalmol. Soc. 51 : 273.

Machemer, R.(1970): Amer. J. Ophthalmol. 69 : 27.

Machemer, R., Norton, E.W.D., Gass, J..D.M. and Chromokos, R. (1970): Amer. J. Ophthal. 71 : 1.

Mathew, M., Mehta, D.K. and Sandhu, P.S. (1979): Ind. J. Ophthal. 27, III : 59.

Michael Shapiro and Suresh, R.C. (1985): Ann. Ophthal. 17 : 231.

Minodo, K. and Kanagami, S. (1976): Fluorography of the fundus periphery with Rhegmatogenous retinal detachment. In: Leey, J.J.D.(ed.). International Symposium on Fluorescein Angiography. Doc. Ophth. Proceeding Series. Dr. W. Junk, B.V. Publishers, The Hague, 1976, Vol. 9 : 573.

Nicholls, J.V.V.(1938): Brit. J. Ophthal. 22 : 672.

Novotny, H.R. and Alvis, D.L. (1961): Circulation, 24:82.

Oberhoff, P., Evans, P.Y. and Delaney, J.P. (1965): Arch. Ophthal. (Chicago), 74 : 77.

O'Day, D., Crock, G. and Galbraith, J.E.K. (1967): Lancet, ii: 224.

Oosterhuis, J.A. and Boen-Tan, R.N. (1971): Ophthalmologica. 162 : 246.

Oosterhuis, J.A. and Lammens, A.J.J. (1965): Ophthalmologica. (Basel), 149 : 210.

Parel, J.M., Crock, G., O'Day, D. and Wigley, A.(1968): Med. J. Aust. 55 : 291.

Parr, J.C., Hodge, J.V., Clemett, R.S. and Knight, P.H. (1968): Trans. Ophthal. Soc. N.Z. 20 : 98.

Parson (1978): Parson's Diseases of the Eye. In: Stephen, J.H. Miller (ed.). FLBS & Churchil Livingstone, Edinburg, 1978, p. 26.

Paul Ehrlich (1881): Quoted from 'Fluorescein Angiography of the Retina'. In: Achim Wessing (ed.). St. Louis, The C.V. Mosby Company, 1969, p. 2.

Pomerantzeff, O.(1975): Invest. Ophthalmol. 14 (5) : 401.

Pomerantzeff, O. and Govingson, J. (1971): Arch. Ophthal. 86 : 420.

Rosen, E.S.(1969): Fluorescence photography of the eye, a manual of dynamic clinical ocular fundus pathology, Butterworth, London, 1969, pp. 23, 27, 102, 126, 125.

Salzmann, M. (1902): Arch. Ophthal. 31 : 41.

Schatz, H., Burton, J.C., Yannuzzi, L.A. and Rabb, M.F. (1978): Interpretation of Fundus Fluorescein Angiography. The C.V. Mosby Company, Saint Louis, 1978, pp. 70, 538. 64, 445, 71.

Shikano, S., Shimizu, K. (1968): Atlas of Fluorescence Fundus Angiography, Tokyo, Igaku Shoin, Philadelphia, W.B. Saunders Co., 1968, pp. 8-10, 78.

Shimizu, K.(1973): Fluorescein Microangiography of the Ocular fundus. Igaku Shoin Ltd., Tokyo, 1973, 45.

Shimizu, K. (1976): A new, TV-Guided Fundus Camera. In: Laey, J.J.D. (Ed.). International Symposium on Fluorescein Angiography, Doc. Ophthal. Proceeding series, Dr. W. Junk B.V. Publishers, The Hague, 1976, Vol. 9 : 37.

Seiker, H.O. and Hickman, J.R. (1953): Circulation, 7 : 79.

Shimizu, K. and Ujii, K. (1976): Fluorescence angiography and angioarchitecture of the choroid. In: Lasy, J.J.D. (ed.), International Symposium on Fluorescein Angiography. Doc.Ophthalm. Proceeding Series. Dr. W. Junk, B.V. Publishers, The Hague, 1976, Vol. 9 : 187.

Shimizu, K., Yokochi, K. and Okano, T. (1974): Jap. J. Ophthal., 18 : 97.

Shimoteri, M. and Aoki, S. (1975): Acta. Soc. Ophthalmol. Jap. 79 : 1748.

Spitznas, M.(1976a): Anatomical correlation of the normal fluorangiography of the fundus. In: Lasy, J.J.D.(ed.). International Symposium on Fluorescein Angiography. Doc. Ophthal. Proceeding Series, Dr. W. Junk, B.V. Publishers, The Hague, 1976, Vol. 9 : 177.

Spitznas, M. (1976b): Peripheral vascular diseases. In: Lasy. J.J.D.(ed.). International Symposium on Fluorescein Angiography. Doc. Ophthal. Proceeding Series, Dr. W. Junk, B.V. Publishers, The Hague, 1976, Vol. 9 : 583.

Stangos, N., Traianidis, P., Papoulis, A., Kambouroglou, J. and Pailas, K. (1976): In: Lasy, J.J.D.(ed.), International Symposium on Fluorescein Angiography. Doc. Ophthal. Proceeding Series, Dr. W. Junk B.V. Publishers, The Hague, 1976, Vol. 9 : 339.

Stein, M.R. and Parker, C.A.(1971): Amer. J. Ophthalmol. 72 : 861.

Talusen, E., Schwartz, Bernard(1977): Arch. Ophthal. 95 : 2166.

Van-Heuven, W.A.J., Schaffer, C.A. and Mehu, M.(1971): Mod. Probl. Ophthal. 9 : 9.

Van-Heuven, W.A.J. and Schaffer, C.A. (1973): Advances in televised fluorescein angiography, Tokyo, 10-14, Igaku Shoin, 1973.

Van-Heuven, W.A.J. and Schaffer, C.A.(1974): International Ophthalmology Clinics, 14 : 31.

Vannas, A. (1969): Acta.Ophthalmologica. Supplementary, 105.

Von-Bayer (1871): Quoted from 'Fluorescein Angiography of the Retina' In: Achim. Wessing (ed.). St. Louis. The C.V. Mosby Company, 1969, p. 2.

Watanabe, C., Yamaji, S. and Yoshihara, M. (1976): Folia Ophthal. Jap. 27 : 645.

Wessing, A. (1969): Fluorescein Angiography of the Retina. St. Louis. The C.V. Mosby Company. 1969. p. 14, 44.

Wessing, A.M.D.(1972): Photocoagulation guided by Fluorescein angiography. In: Shimizu, K. (ed.), Fluorescein Angiography Proc. Int. Symp. on Fluorescein Angiography (ISFA). Tokyo, 1972 : 410, Igaku Shoin Ltd. , Tokyo, 1974.

Wessing, A. and Schlicke, B. (1976): Equatorial degenerations with atypical vessels. In: Leavy, J.J.D.(ed.). Int. Symp. on Fluorescein Angiography. Doc. Ophthal. Proceeding Series, Dr. W. Junk, B.V. Publishers, The Hague, 1976, Vol. 9 : 613.

Wyber, K.C. (1954): Brit. J. Ophthal. 38 : 513.

Wyber, K.C. (1956): Brit. J. Ophthal. 40 : 65.

Yoshihara, H. (1979): Acta. Soc. Ophthal. Jap., 82 : 610.

Yoshihara, M., Yamaji and Yamamowa, K. (1976): Folia Ophthal. Jap. 27 : 361.

Yuhasts, Z., Akeshi, R.H., Urban, J.C. and Mueller, M.M.M. (1973): Arch. Ophthal. (Chicago), 90 : 481.

APPENDIX

PROFORMA FOR RECORDING EACH CASE INDIVIDUALLY FLUORESCEIN ANGIOGRAPHY OF POSTERIOR FUNDUS IN MYOPIA

Case No. OPD/MRD No.

Film No. PIB No.

Name of patient Age/Sex

Address

Family History of Myopia Present/Absent

Brief History of complaints

I. Visual Acuity RE LE

(a) Uncorrected

(b) Corrected

II. Refraction under

Drosyn/H.A./Atropine

III. Post Mydriatic Test

IV. General Ocular Examination

(i) Eye ball

(ii) Lids

(iii) Conjunctiva

(iv) Cornea

(v) Anterior Chamber

(vi) Iris

(vii) Pupil

(viii) Lens

(ix) Tension

V. Slit Lamp Examination

-Cornea

-Anterior Chamber

-Lens

-Vitreous

VI. Direct Ophthalmoscopic Examination

RE LE

-Media

-Optic disc

-Colour & Shape

-Margins

-Cupping

-Crescent

-Blood Vessels:

-A.V. Ratio

-Arteries

-A/V Crossing

-Veins

-Haemorrhages

-Exudates

-Foveal Reflex:

-Macula

-General fundus picture

VII. Fluorescein Angiographic Report:

-Gross Angiographic changes

RE LE

-Early Arterial phase

-Arterio venous phase

-Late venous phase